

# JOURNAL of the American Veterinary Medical Association

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The American Veterinary Medical Association

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# **JOURNAL**

*of the*

## **American Veterinary Medical Association**

*Formerly AMERICAN VETERINARY REVIEW*

(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 1230 W. Washington Blvd., Chicago, Ill.

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### **PROBLEMS FOR OUR COLLEGES**

Considering the fact that hundreds of letters are received at the A. V. M. A. office every month, it is not surprising that some of these should be of more than passing interest. No small number of these letters are written by comparatively recent graduates asking for advice or information. Occasionally one of these registers a mild complaint that the veterinary profession is not what it ought to be or is not what was represented in a college catalog or prospectus. Not infrequently letters have contained rather severe criticisms of the veterinary curriculum. In each case, the criticism, of course, has been directed mainly against the college attended by the writer of the letter, not in any spirit of disloyalty, but as a plain statement of fact.

The complaint most frequently made is that too little attention has been given to some of the practical sides of general practice. This problem is not a new one. It is as old as veterinary education itself. There is serious question whether it is possible to teach some of these things within the college walls. The ambulatory clinics which have been developed at some of our veterinary colleges have helped, in part at least, to fill the void. The suggestion has been made that it would be advisable to require every veterinary student to spend a prescribed amount of time with a practitioner as part of his course. This was the custom at the

Ontario Veterinary College, when that institution maintained a two-year course. Each student was required to spend the vacation between the first and second years with a preceptor, as a requirement for graduation.

There is another part of the training of veterinary students that appears to be neglected, at least in some of the colleges. Too little attention is given to such things as business methods, professional ethics, hospital management, case records, and many of the finer details, that contribute to making a successful practitioner. If these things were not being neglected there would be fewer evidences of the fact.

This morning a veterinarian's check came to our attention. It was signed "Dr. X. Y. Jones." Had this man been taught that it is considered bad form or poor taste to use "Dr." in connection with his signature on a check? Several days ago a newspaper clipping was received, containing the announcement of a 1932 graduate opening an office in a midwestern city. It was anything but professional in character. Had this veterinarian been taught anything as to the proper method of announcing his presence in a new location? Here is a letter from an established practitioner needing an assistant and wanting to be put in touch with a young graduate. But he took pains to say that he did not want a graduate of a certain institution because "they teach nothing but theory there." Here is a letter from a recent graduate asking whether it would be "all right" for him to handle a certain brand of canned dog food "as a side-line." These are just a few examples. They are suggestive of many others of a related character. Separately many appear trivial. Collectively they constitute something for our educators to think about.

#### **AFFILIATION PROPOSAL RATIFIED**

At the December meeting of the Massachusetts Veterinary Association the plan for affiliation with the American Veterinary Medical Association was approved, and similar action was taken by the Ohio State Veterinary Medical Association at the annual meeting held in Columbus on January 11-12. Ohio, therefore, was the thirtieth state to approve the proposal, thereby clearing the way for putting the plan of affiliation into effect, the A. V. M. A. previously having voted to do this just as soon as possible after thirty associations had ratified the proposal.

Unofficial information has been received to the effect that the Wisconsin Veterinary Medical Association approved the plan at the annual meeting held January 10-11, and that similar action had been taken by the Tennessee Veterinary Medical Association

at the meeting held January 23-24. The action taken by these organizations brings the number up to thirty-two, leaving only fifteen state associations to take action. Mississippi and Nevada have already indicated that the plan would be approved at the first opportunity.

The thirty-two states are:

California	Massachusetts	Oklahoma
Colorado	Michigan	Oregon
Connecticut	Minnesota	Pennsylvania
Florida	Missouri	South Carolina
Georgia	Montana	Tennessee
Illinois	Nebraska	Texas
Kansas	New Jersey	Utah
Kentucky	New York	Vermont
Louisiana	North Carolina	Virginia
Maine	North Dakota	Wisconsin
Maryland	Ohio	

Several amendments to the Constitution and By-laws of the A. V. M. A. were prepared and submitted at the meeting in Atlanta, and these will come up for action at the meeting in Chicago this summer. Assuming that the amendments will be approved, the next step will be to perfect plans for organizing the House of Representatives which will function for the first time at the meeting in New York in 1934.

There has been no desire upon the part of the A. V. M. A. to force any state or province to take favorable action on the plan for affiliation. It probably would be safe to say that in most of the cases where no action of any kind has been taken, either for or against, the reason could be found in a lack of understanding of what the plan is intended to accomplish. This matter is being brought to the attention of our members in those states and provinces which have taken no action, so that they may know just what the present status is. As the matter now stands, with the plan approved, it means simply that those organizations which have not voted to affiliate will have no voice or vote in the House of Representatives when it is organized next year, unless favorable action is taken in the meantime.

### DOCTOR FLYNN ELECTED

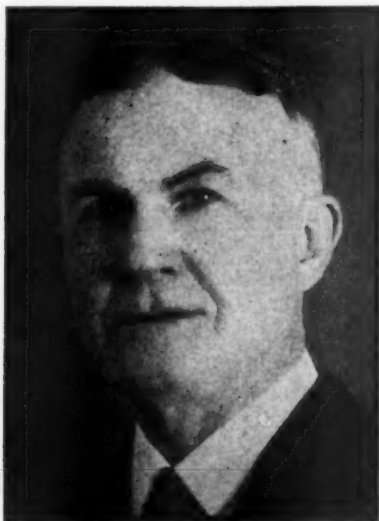
The special election held in Executive Board District 8 came to a close on January 7 and a canvass of the ballots cast indicated the election of Dr. J. C. Flynn, of Kansas City, Mo., for the balance of the unexpired term of Dr. N. F. Williams, of Fort Worth, Texas, who was elected to the presidency at the meeting in Atlanta last summer. Dr. Flynn needs no introduction to the profession in America. During the past ten years

or more he has appeared on the programs of numerous veterinary meetings in all parts of the United States and has attended most of the meetings of the A.V.M.A. held since he has been a member.

Dr. Williams continues as an ex-officio member of the Executive Board until the close of the 1934 meeting.

### VICE-PRESIDENT AXBY DIES

Death loves a shining mark. Dr. W. A. Axby, of Harrison, Ohio, third vice-president of the American Veterinary Medical Association, eminent veterinarian, prominent Kiwanian and sterling citizen, died at Christ Hospital, Cincinnati, Ohio, Jan-



DR. W. A. AXBY

uary 20, 1933, after a brief illness due to septic sore throat. Funeral services were held at Harrison, on January 22, with members of the profession from all parts of Ohio, Indiana and Kentucky in attendance. A biographical sketch and further appreciation of this outstanding veterinarian will be published in the March issue of the JOURNAL.

### OLDEST MEMBER DIES

Just as the forms for this issue of the JOURNAL are being closed, we learn of the death of Dr. John C. Meyer, of Cincinnati, Ohio, the oldest member of the A. V. M. A. He had been a member in good standing since 1875.

## APPLICATIONS FOR MEMBERSHIP

(See January, 1933, JOURNAL)

### FIRST LISTING

- JAFFRAY, JOHN B. 2956 Washington Blvd., Chicago, Ill.  
M. D. C., Chicago Veterinary College, 1903  
Vouchers: David S. Jaffray and H. Preston Hoskins.
- KENDALL, GORDON C. 3-4 War Memorial Bldg., Nashville, Tenn.  
D. V. M., Alabama Polytechnic Institute, 1928  
Vouchers: M. Jacob and G. A. Metcalf.
- MCCUNE, EUGENE F. 7401 S. Puget Sound Ave., Tacoma, Wash.  
B. S., D. V. M., Washington State College, 1931  
Vouchers: R. A. Button and J. E. McCoy.
- NOLAN, JAMES D. Box 912, Raymond, Wash.  
B. S., D. V. M., Washington State College, 1932  
Vouchers: E. E. Wegner and G. W. McNutt.
- SWEENEY, MARTIN A. 6523 South Park Ave., Chicago, Ill.  
D. V. M., Chicago Veterinary College, 1914  
Vouchers: L. A. Merillat and E. L. Quitman.

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### Applications Pending

#### SECOND LISTING

(See January, 1933, JOURNAL)

- Glueck, Oscar, Blue Point, L. I., N. Y.  
Purmell, Louis C., 1810 First Ave., New York, N. Y.

The amount which should accompany an application filed this month is \$9.58, which covers membership fee and dues to January 1, 1934, including subscription to the JOURNAL.

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## COMING VETERINARY MEETINGS

- Connecticut Veterinary Medical Association. Hartford, Conn.  
February 1, 1933. Dr. Edwin Laitinen, Secretary, 993 N.  
Main St., West Hartford, Conn.
- New York City, Veterinary Medical Association of. Academy  
of Medicine, 5th Ave. and 103rd St., New York, N. Y. Feb-  
ruary 1, 1933. Dr. John E. Crawford, Secretary, 708 Beach  
19th St., Far Rockaway, Long Island, N. Y.
- San Diego-Imperial Veterinary Medical Association. San Diego,  
Calif. February 1, 1933. Dr. A. P. Immenschuh, Secretary,  
Santee, Calif.
- Alabama Veterinary Medical Association and Short Course for  
Graduate Veterinarians. College of Veterinary Medicine, Ala-  
bama Polytechnic Institute, Auburn, Ala. February 7-11, 1933.  
Dr. C. A. Cary, Dean, Alabama Polytechnic Institute, Auburn,  
Ala.

- Hudson Valley Veterinary Medical Society. Albany, N. Y. February 8, 1933. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.
- Southeastern Michigan Veterinary Medical Association. Detroit, Mich. February 8, 1933. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.
- Tulsa County Veterinary Association. Tulsa, Okla. February 9, 1933. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.
- Interstate Veterinary Medical Association. Elks Bldg., Omaha, Neb. February 13, 1933. Dr. G. L. Taylor, Secretary, Plattsmouth, Neb.
- Chicago Veterinary Medical Association. Hotel LaSalle, Chicago. February 14, 1933. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.
- Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. February 15, 1933. Dr. E. E. Jones, Secretary, 1451 Mirasol St., Los Angeles, Calif.
- Illinois State Veterinary Medical Association. Saint Nicholas Hotel, Springfield, Ill. February 15-16, 1933. Dr. D. F. Luckey, Secretary, Hotel Silas, Springfield, Ill.
- Northwestern Ohio Veterinary Medical Association. Hotel Waldorf, Toledo, Ohio. Dr. W. P. S. Hall, Secretary, Division of Health, 9 Ontario St., Toledo, Ohio.
- Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. February 21, 1933. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.
- Keystone Veterinary Medical Association. Philadelphia, Pa. February 22, 1933. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.
- Louisiana State University Short Course for Veterinarians. Baton Rouge, La. March 1-2, 1933. Dr. Harry Morris, Assoc. Prof. Veterinary Science, Louisiana State University, Baton Rouge, La.
- East Tennessee Veterinary Medical Society. White Surgical Supply Co., Knoxville, Tenn. March 11, 1933. Dr. R. E. Baker, Secretary, Morristown, Tenn.
- American Veterinary Medical Association. Palmer House, Chicago, Ill. August 14-18, 1933. Dr. H. Preston Hoskins, Secretary, 1230 W. Washington Blvd., Chicago, Ill.

## HOW CAN THE OPERATION OF THE ARMY VETERINARY SERVICE BE IMPROVED IN FUTURE ENGAGEMENTS IN THE LIGHT OF EXPERIENCES OF THE WORLD WAR?\*

*By Major R. A. KELSER, V. C., U. S. Army  
Army Medical School, Army Medical Center  
Washington, D. C.*

On April 6, 1917, the late President Woodrow Wilson proclaimed to the world the Resolution of the United States Congress "that the state of war between the United States and the Imperial German Government which has been thrust upon the United States is hereby formally declared."

With that historical pronouncement came the pledge of the great wealth and vast resources of the country to bring the war to a speedy and successful conclusion. Money was soon pouring into the treasury in staggering amounts, essential industry of all sorts leaped into mass production, farmers of the nation moved to increase vastly the supply of basic foodstuffs, patriotic fervor ran high and the great masses of loyal Americans were anxious to help. However, in spite of all of that, it is a well-known fact that, from a strictly military standpoint, in so far as concerned adequate preparedness *promptly* to undertake the effective prosecution of the war, we were far short of the requirements.

It became necessary to develop and put into effect on short notice plans for the recruiting, mobilization, equipment, feeding, housing and training of an army of a size hitherto unknown to the country. Then came the many problems of transportation of men, animals, supplies and equipment; manufacture of munitions; ship-building; production and regulation of food supplies; all constituting a mammoth task long to be remembered. No small problem in this great movement was the drafting and assembling of large numbers of horses and mules to meet military demands. With this mobilization of animals came the problems incident to the care of their health. While, as already indicated, the Army as a whole was not adequately prepared to meet promptly the situation which confronted it, it can be stated, without fear of contradiction, that no element of the military establishment was less prepared than the veterinary service. Let

\*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

it be said at once, however, that the veterinarians in the service at the time of declaration of war were in no way responsible for this situation.

A Congressional Act, approved June 3, 1916, gave rank to Army veterinarians and provided for a Veterinary Corps in the Medical Department, consolidating, in such corps, veterinarians of cavalry and field artillery, quartermaster veterinarians and veterinary inspectors of animals. This Act made possible a relatively small corps of veterinary officers in the grades of second lieutenant to major, inclusive. While the Act was approved June 3, 1916, the routine examinations and other details incident to putting the legislation in effect delayed the actual commissioning of the first of these veterinarians until some time in March, 1917. By April 11, 1917, a total of only 62 veterinarians had qualified and were commissioned in the regular establishment and at the beginning of the fiscal year (July 1, 1917) there were but 91 veterinary officers in the Regular Army. Thus, there was no organized, commissioned Veterinary Corps in operation at the time the United States entered the World War.

#### VETERINARY ADVISORY COMMITTEE

In June, 1917, Major Gerald E. Griffin (now Colonel Griffin, retired), who was then the senior veterinary officer of the Army, was ordered to Washington for duty. Prior to that time the Corps was not represented by a Veterinary Officer in the War Department at Washington. Shortly following Col. Griffin's assignment, the Surgeon General invited to Washington several prominent civilian veterinarians to serve as a Veterinary Advisory Committee in connection with the development of a veterinary service capable of meeting the demands of war. Several members of this committee later were commissioned in the Veterinary Corps and served throughout the period of hostilities.

On July 14, 1917, the Surgeon General of the Army submitted to the Adjutant General a proposed veterinary organization plan developed by the Advisory Board. This plan contemplated 1 veterinary officer and 16 enlisted men for every 400 animals, the officers to be commissioned in grades corresponding to those in the Medical Corps.

On October 4, 1917, the War Department published General Orders 130, establishing the Veterinary Corps, National Army, and approved the ratio of officers and enlisted men which the Advisory Board had recommended but limited the highest rank of officers to that of major. Subsequently, however, 2 colonels

and 6 lieutenant colonels were authorized. It might be said here that this ratio of veterinary officers to animals (2.5 officers per 1,000 animals) soon proved inadequate and was gradually increased to 4.7 officers per 1,000 animals.

Following this authority for the establishment of the Veterinary Corps, plans for its organization, development and operation were promptly made and put into execution in the United States. Special Regulations 70, governing the Army veterinary service, were promulgated and approved by the Secretary of War on December 15, 1917.

#### DIRECTOR OF THE VETERINARY CORPS

Under Regulations 70 responsibility for the administration of the veterinary service was vested in a Director of the Veterinary Corps serving directly under the Surgeon General. This officer and his staff constituted an important division of the Surgeon General's Office. The position of Director of the Veterinary Corps was held for two periods during the war and for several years after the close of hostilities by officers of the Medical Corps.

By October 1, 1917, a total of 795 veterinary officers were on active duty. A large percentage of these officers were commissioned direct from civil life. A few came in with National Guard organizations and a few more were commissioned from the Veterinary Section of the Medical Enlisted Reserve Corps. It will be recalled that it was possible to enlist veterinary students of draft age, who were at the time in attendance at recognized veterinary colleges, maintain them on inactive status within the Medical Enlisted Reserve Corps until graduation and then call them to active duty. This arrangement actually supplied only a few veterinary officers for active duty because the war ended before the flow of officers from this source had time to get well under way. However, the plan was an excellent one and in due time would have constituted the chief source of supply of veterinarians for the Army. The largest number of veterinary officers on duty at any time was 2,234, on November 30, 1918.

In the United States (Service of the Interior) the veterinary service was organized to provide (1) veterinary service in connection with the purchase and transportation of animals; (2) veterinary service at remount depots; (3) veterinary service at posts, in camps, and on the march; (4) veterinary service at embarkation depots and on transports; (5) veterinary service at subsistence depots; (6) veterinary laboratory service; (7) veterinary supply divisions in medical supply depots.

Because of the lack of an organized Veterinary Corps at the start of the war, the problem of training both commissioned and enlisted veterinary personnel constituted an even more important problem in our service than in some of the other branches. To meet this need, training was instituted at a number of places, the more important being the Veterinary Section of the Medical Officers Training Camp at Camp Greenleaf, Ga., and the Veterinary Training School at Camp Lee, Va. A school for training officers in meat and forage inspection was established in Chicago, and in Philadelphia officers who were to be assigned veterinary laboratory work were given instruction at an Army Veterinary Laboratory which had been organized at the University of Pennsylvania.

#### REMOUNT DEPOT AT EACH CANTONMENT

To accommodate animals mobilized in the United States, a remount depot was constructed in connection with each of the large divisional cantonments. At each of these remount depots veterinary hospital facilities of a very unsatisfactory type were provided. The remount depots became overcrowded and, with the limited space and poor facilities for the isolation and handling of sick animals, it was extremely difficult to control communicable diseases satisfactorily. Yet, under such circumstances, the vast percentage of veterinary hospital work in the United States during the war was conducted at these remount depots. The veterinary hospital service at the camp remount depots was carried out by units consisting of from 6 officers and 75 enlisted men, for the smallest depots, to 12 officers and 150 men, for the largest depots.

The error of establishing veterinary hospital facilities at remount depots occurred both in the United States and France and in so far as concerned the animal situation was one of the most serious mistakes made. Certainly in any future engagement this is one error that will be avoided.

Divisional veterinary service in the camps proper was administered by a Division Veterinarian. This officer bore the same relationship to the Division Commander, in veterinary matters, as the Division Surgeon did in medical matters. Sick animals of a division, requiring hospitalization, were evacuated to the veterinary hospital of the adjacent remount depot.

After a division left its cantonment for overseas service, the veterinary service of the camp was conducted by a Camp Veteri-

narian with such commissioned and enlisted personnel as was required.

The purchase of animals for the Army was made by purchasing boards to each of which was assigned a veterinary officer.

With a view to arranging for the shipment of animals overseas, animal embarkation depots were organized at Newport News, Va., Charleston, S. C., and Hoboken, N. J. The veterinary service at these ports was administered by a Port Veterinarian. Veterinary service on animal transports was conducted by Transport Veterinarians, operating under the Port Veterinarian of the port of embarkation.

Veterinary laboratory service was provided by a large Army Veterinary Laboratory operating in Philadelphia, Pa., and by assigning a veterinary officer to each of the laboratories in the various military departments into which the country then was divided.

#### INSPECTION OF MEAT AND DAIRY PRODUCTS

An important phase of the veterinary service in the United States during the war was the inspection of meat, meat-food and dairy products purchased for the Army. In the beginning of the war, considerable confusion existed in getting this service into satisfactory operation. This was due largely to the fact that these inspections were being carried out at a number of places by lay inspectors of the United States Bureau of Animal Industry, assigned at the request of the Quartermaster Corps; by Army veterinary officers at other points, and in some instances by medical sanitary officers. In some cases no inspections of any sort were made. This situation, however, was cleared up at a conference at the Surgeon General's Office between representatives of the Bureau of Animal Industry, the Public Health Service, the Veterinary Corps and the Quartermaster Corps. It was agreed at this meeting that the Army Veterinary Corps should take over exclusively the operation of this service. Proper regulations were promulgated accordingly and the service went forward in an entirely satisfactory manner.

This food inspection service is an important function of the Army Veterinary Corps and under war conditions it must be greatly expanded. Thus, in connection with war procurement plans, for future reference, such service demands careful consideration.

With a view to supervising and coördinating the work of the veterinary service in the field in the United States, the country was divided into five zones and the operation of the service in

each zone placed under the observation of a Veterinary General Inspector. This officer kept the Surgeon General's Office fully advised at all times as to the status of veterinary affairs in his particular zone.

While the organization and operation of the veterinary service in the United States was far from perfect, in so far as any comparison can be made under the markedly different conditions that existed in the United States and overseas, the service at home was much more satisfactory than that abroad.

When the first contingent of the A. E. F. arrived in France, it included no veterinary organization and little thought was given to such a service. Back in December, 1915, William P. Hill (now Col. Hill), an Army veterinarian, was ordered to France as a military observer. He served in such capacity until the United States declared war. At that time he joined the Chief of the remount service in France for the purpose of buying horses from the French for the use of our troops on their arrival in the theater of operations. Hill's experience, of course, had impressed upon him the importance and necessity of providing adequate veterinary service for the American Army but his suggestions and recommendations, early made, were given little heed. Major Hill was later (April 3, 1918) made Chief Veterinarian, which position he held for about four months, when he was relieved by Col. David S. White.

#### ORGANIZATION PLAN APPROVED

The first provision of any sort for a veterinary service in the A. E. F. was contained in a general organization plan approved by the Commander-in-Chief of the expeditionary forces on July 10, 1917. This plan provided for a mobile veterinary hospital for each Corps and Army. Such hospital units were to be operated by 4 officers and 150 enlisted men. This meager, inadequate provision for veterinary service resulted in a cable being sent to the Commander-in-Chief of the A. E. F. by the War Department conveying the recommendations of the Surgeon General for a more adequate service based on animal strength and probable percentage of incapacity. This advice from the War Department brought forth a statement from the Commander-in-Chief that in addition to the plan already made for the veterinary service of each Corps and Army, he was proceeding with the organization of veterinary facilities in the service of the rear, but the War Department suggestions did not result in any material change

in the A. E. F. plan to keep the veterinary service attached to the remount branch.

Although the Veterinary Corps legally belonged to the Medical Department of the Army, in the A. E. F. it was organized and operated for nearly a year under the remount service of the Quartermaster Corps. Realizing that the Veterinary Corps would undoubtedly fail to perform its mission satisfactorily, because of the circumstances under which it was being organized and operated at the time, the Surgeon General, in November, 1917, dispatched to France Major L. A. Klein and Lieut. A. L. Mason (now Col. Mason) for the purpose of consulting with the General Staff of the A. E. F. in connection with the organization and equipment of our overseas veterinary service.

#### RECOMMENDATIONS MADE TO CHIEF SURGEON

These two officers, in the face of many trials and difficulties, made a careful survey of the situation and later in December, 1917, submitted a comprehensive report to the Chief Surgeon of the A. E. F., setting forth recommendations for the organization and operation of the Veterinary Corps as contemplated by War Department General Orders 130 and Special Regulations 70.

Supporting the efforts of Klein and Mason to fulfill their mission, came letters and cablegrams from the Surgeon General of the Army in Washington to General Pershing and to the Chief Surgeon of our Army in France. On January 5, 1918, the Surgeon General wrote the Chief Surgeon a letter, from which the following is quoted:

The Medical Department is charged by law with the responsibility for the administration of the veterinary service, and it is believed that this responsibility can not be evaded. The department, therefore, does not approve, for the present, the amalgamation of the veterinary service with any other branch of the military service.

On January 21, 1918, the Surgeon General sent a cablegram to General Pershing, from which the following is extracted:

Veterinary service in United States reorganized and placed on independent, sound working basis suitable to requirements modern warfare. Principle followed similar to British service, excepting it is under direction of Surgeon General, which change now recommended by British. Suggest immediate steps be taken to similarly organize veterinary service with American Expeditionary Forces, creating chief veterinarian, and vesting in him direct control and responsibility to chief surgeon and commanding general.

In spite of all of their efforts and the support from Washington, the mission of Klein and Mason for the time being was doomed to failure, as the order of the Commander-in-Chief of the

A. E. F. suspending so much of War Department orders and Regulations 70 as placed the Veterinary Corps under the direct supervision of the Medical Department remained in force and the service continued for eight months more under the remount service.

Because of the shortage of shipping facilities for troops, it was decided early in the war to defer the shipment of animals from the United States and to buy them abroad. The French agreed to provide 7,000 horses monthly. However, in August, 1917, just a short time after this agreement was made, they advised that it would be impossible to furnish this number. Hence the War Department at Washington was confronted with the proposition of providing animals from this country.

#### SHORTAGE OF HORSES IN FRANCE

The acute shortage of ship tonnage and the plan to push the transportation of men to the limit made it impossible to meet our own animal needs in France, even though we had assembled a plentiful supply of horses and mules in the United States. Thus, in April, 1918, a year after the declaration of war, and with six American divisions in France, we had with them a total of only 55,378 animals, including those in remount depots. On November 1, 1918, practically at the close of the war, our animal shortage in France exceeded 100,000, a figure equal to about one-half of estimated needs. At one time, General Pershing, through a strenuous personal effort, succeeded in having the French Government supply the American forces with 50,000 animals. Again, in the face of an acute situation during the Meuse-Argonne drive, Marshall Foch provided 13,000 animals for the use of the A. E. F.

During the war a total of only 66,071 horses and mules, a little better than 25 per cent of our estimated requirements, was shipped to our forces in France. These shipments were made between October 15, 1917, and November 30, 1918.

It goes without saying that with the horse shortage in France such animals as we could obtain locally were far from the best. It was a case of take anything that could be had, irrespective of condition or price. A horse was a horse and if he was capable of movement, whether or not he was diseased was of little concern to purchasing officers.

Failure to ship to France a sufficient number of animals to meet the needs of our own organizations was another serious mistake. True, the cry was for men, but unless we were to supply men merely as replacements for foreign armies, then, under the condi-

tions that existed, it was as important that we supply our troops with their animal complement as it was that they be supplied with any other essential equipment.

There was no satisfactory coordination whatever of veterinary activities in our overseas Army. Conflicting assignments were made in veterinary personnel. It frequently happened that orders issued at one place, to be carried out by the veterinary personnel of another locality, could not possibly be complied with. Lack of sufficient authority to act in various administrative matters pertaining to veterinary service commonly resulted in the necessity of going through long, time-consuming channels to handle situations which required immediate action.

The lack of proper organization, with the attendant utter inadequacy of hospital facilities and equipment, means for prompt and efficient evacuation of animals, provisions for medical supplies, and the proper distribution and control of veterinary personnel, resulted in a state of affairs greatly to be regretted. Mange, glanders, ulcerative lymphangitis and other infectious diseases became rampant and whole organizations were threatened with immobility as a result of being "dehorsed." By July 1, 1918, the sick-rate among the animals of the A. E. F. had reached an enormous proportion.

#### STORY TOLD BY DOCTOR MERILLAT

To go into the details of even a part of the various important factors and incidents which operated to the detriment and chagrin of the veterinary service in France during this period would not only be depressing but would require far more time than is at my disposal. To those who are especially interested in this phase of the subject I commend the serial now running in the *North American Veterinarian*, entitled "The Veterinary Service of the A. E. F.," by Dr. L. A. Merillat, former Chief Veterinarian, First Army, A. E. F. Suffice it to say that, largely as a result of factors over which responsible veterinarians in France and the Surgeon General's Office and War Department in Washington had no control, the veterinary service of the A. E. F. was prevented from rendering the type of service of which the profession was capable.

As the war progressed into the summer of 1918 and the shortage of animals became more and more acute, the importance of veterinary service and the seriousness of its failure to function efficiently began to become evident to the General Staff at A. E. F. Headquarters. Then it was, at such late date, that serious attention was directed towards this service.

On July 3, 1918, General Pershing cabled the War Department to send him the best available veterinarian for administrative duty. As a result of this request, Col. D. S. White sailed for France on July 30, 1918. Under date of July 26, 1918, before Col. White's arrival, General Pershing revoked his early order by which he had stood all of this time and directed that the Veterinary Corps now be reorganized and operated as a part of the Medical Department.

The decision to reorganize the veterinary service under the Medical Department, while obviously an important step towards the correction of a bad situation, could not be expected to cure all of the ills of the Veterinary Corps overnight. Further, certain factors beyond the control of the Medical Department operated to the detriment of the veterinary service and resulted in the replacement of the Chief Veterinarian with an officer of the mounted service who was not a veterinarian. The war ended in a little over two months after the veterinary service was transferred to the Medical Department. It was thus impossible in this brief period for the Veterinary Corps to correct its organization and prove its merits.

#### SOUND ADVICE WENT UNHEEDED

From the data presented I have endeavored to indicate that the poor showing of the Veterinary Corps in France was primarily due to the failure of responsible line officers of the American Expeditionary Forces early to recognize the importance of an adequate, well-organized veterinary service and their unwillingness to concede the necessity of such a service, organized and operated as recommended by those capable of giving sound advice on the subject.

The question naturally arises as to why this attitude prevailed at A. E. F. Headquarters. It will be recalled that when the first contingents of our Army went overseas the Headquarters of the A. E. F. was composed, to a large extent, of officers who had served with General Pershing in his Mexican expedition. In the Mexican project, as well as in all previous engagements in which the United States was involved, the animal supply available to the Army was always vastly greater than the need. Thus, when we went into the World War our Headquarters in France undoubtedly believed that it would be entirely possible and more satisfactory to replace disabled animals than to salvage them. Under such circumstances veterinary service would not be considered particularly important. However, when the unlimited animal

supply was not forthcoming, the need for veterinary service was great but unfortunately it had not been adequately provided for.

In the light of the experiences of the World War, what assurances have we that a future emergency might not witness a similar situation as regards Army veterinary service? In this respect we are optimistic and have ample reason for being so.

In the first place the costly lessons of the past war, we hope, will serve to prevent a duplication of many mistakes and errors. Then, for the first time in the history of our country, the United States has what should prove to be fairly adequate war plans for a major emergency. In these plans the veterinary service has been considered as an important and essential cog in the war machine and ample provision has been made for the organization and operation of such service.

#### THE ARMY VETERINARY CORPS

Our present Regular Army Veterinary Corps, while small, is a well-organized, efficient unit of the military establishment. It has been alert to its responsibilities and has constantly endeavored to develop and improve its service. We have developed a Veterinary School of the highest order for the professional and technical instruction of veterinary officers and enlisted personnel. Our Medical Field Service School at Carlisle, Pa., is a model institution for the training of Medical, Dental and Veterinary personnel in field duties. Should the occasion arise, we are confident that our regular Corps will fully measure up to requirements in serving as an efficient, well-organized, smooth-functioning nucleus, around which a service sufficient for war needs of a major undertaking readily can be built.

While we are confident that with our present organization and the provisions made for its expansion in time of war, we would not be likely to witness a repetition of the break-down of 1917-18, constant attention must be given to keep this service abreast of changing conditions and policies.

Ardent advocates of increased mechanization of the Army have advanced the thought that with such mechanization veterinary service can be reduced materially. As a matter of fact, the present authorized strength of the Corps represents an irreducible number, if our service is properly to perform its varied duties which are not alone limited to the actual care of sick animals.

While, as indicated, we consider present organization and war expansion plans reasonably adequate, we must see to it that

accomplishments of years are not torn down. The present-day tendency towards mechanization might well be pushed by enthusiastic supporters to the point where it would endanger adequate, effective military veterinary service in both peace and war periods. The horse is not out of the war. Wherever we put troops and implements of war we must also put transportation. While mechanized transportation has its place and should most assuredly be made use of, the time has not arrived when the machine can satisfactorily traverse the terrain beset with mountain trails and ravines, heavily wooded areas, rivers and streams. Nor can the machine push on for another vital half-mile with an empty gas tank or a broken part. The horse can, and it is because of these circumstances that he is still an indispensable factor in modern armies.

#### VETERINARY OFFICERS SHOULD HAVE AUTHORITY

In time of war the Veterinary Corps should be given every opportunity to organize, develop and administer its service in accordance with war plans that have been carefully worked out after much study and thought by capable individuals. While properly under the jurisdiction of the Medical Department, the veterinary branch should be given sufficient latitude of authority and such freedom of action as will permit it to perform its mission effectively. Thus, after having placed carefully selected veterinary officers in the positions of highest responsibility, they should have sufficient rank, authority, freedom of action and confidence of responsible troop commanders to enable them to carry out their task effectively. To charge veterinary officers in key positions with professional responsibilities and yet deny them sufficient authority to organize, coördinate and administer their service properly is a tragedy we hope will never be repeated.

The veterinary profession of the United States has a definite responsibility in the matter of veterinary military service. The responsibility of serving the country in a professional way in time of war is just as great as that of the layman called upon actually to bear arms. Thus, in any great national emergency the obligation for adequate, efficient veterinary service with our armies rests upon the profession as a whole and not upon any one group. The regular Corps is authorized but 126 officers and in time of great stress would serve as only a nucleus for the force actually needed. It behooves our entire professional membership to lend all possible support to our Corps in its efforts to maintain an adequate, efficient peace-time organization capable of being

expanded promptly and effectively, to meet the needs of war should the occasion unfortunately arise.

As I have indicated, in the development of war plans for the Armies of the United States the importance of veterinary service has been recognized and adequately provided for. These plans call for 1,647 veterinary reserve officers. On July 31, 1932, we had but 1,053 in our Reserve Corps. This is a shortage of nearly 600 officers. With recent legislation operating to abolish R. O. T. C. units at medical, dental and veterinary colleges, a potent source of young reserve officers will be lost. Thus, a step that the veterinary profession could readily take to improve our preparedness for service, should war necessitate it, would be to wipe out the deficit that exists in our Veterinary Reserve Corps.

### Horsetail No Remedy for Diabetes

Interstate sale of a product composed of the ground, sterile branches of field horsetail (*Equisetum arvense*), labeled as a relief for diabetes and anemia, cost Charles F. Diller, trading as the Photo-Synthetic Tea Company, Lancaster, Pa., a fine of \$25. This "photo-synthetic tea," so-called, according to the U. S. Department of Agriculture, was labeled in part as follows: "Relieves diabetes and prevents anemia by making the sugar normal and the blood red." The government held that the labeling was false and fraudulent under the federal Food and Drugs Act, and the federal court for the Eastern District of Pennsylvania, upon a plea of *nolo contendere* by the defendant, assessed the fine. The herb, *Equisetum*, has been tried out for diabetes at hospitals and found to be worthless in the treatment of the disease. The goods had been shipped from Lancaster to Lawrence, Mass.

Recent action by the government in removing from interstate commerce a worthless diabetes "remedy" brings again to the attention of the public the fact that in the 26 years of enforcing the Food and Drugs Act, officials have taken similar action against scores of useless diabetes preparations. Such widely different products as almond and bran bread, suprarenal extracts, epinephrine, opium, sodium bicarbonate, levulose—and now, common field horsetail—have been falsely and fraudulently labeled with remedial claims for this serious malady. There is no drug nor combination of drugs known to medical science which can cure the disease.

All dogs in DeKalb County, Tennessee, were placed under quarantine for ninety days, effective January 14, 1933, following an order announced by Dr. J. M. Jones, State Veterinarian.

## STUDIES ON CANINE DISTEMPER:

### III. A Comparison of Natural and Experimental Virus Infections\*

By A. S. SCHLINGMAN, *Detroit, Michigan*

*Research and Biological Laboratories*

*Parke, Davis and Company*

Distemper of dogs has been known and recognized as a contagious disease since the latter part of the eighteenth century. A variety of organisms were advanced by early investigators as the cause of this malady but it was not until 1905 that Carré<sup>1</sup> reported a filtrable virus as the etiological agent. This work, later confirmed by Lignieres,<sup>2</sup> was not in keeping with the results of Ferry,<sup>3,4</sup> M'Gowan,<sup>5</sup> Torrey and Rahe<sup>6</sup> and Schoiche,<sup>7</sup> who concluded from studies of naturally infected cases that a micro-organism (*Bacillus bronchisepticus*) was the causative factor. More recent work of Carré,<sup>8</sup> Puntoni<sup>9</sup> and Dunkin and Laidlaw<sup>10</sup> indicated that they were working with a filtrable virus disease.

Studies of naturally infected cases obtained in the vicinity of Detroit, Michigan, by the author<sup>11</sup> during the past few years showed that the causative agent in the majority of these cases was *B. bronchisepticus*. Injection of ferrets with sterile filtrates of suspensions of spleens from these affected animals did not show the presence of a filtrable virus,<sup>12</sup> such as had been demonstrated by other workers.

It would seem from the reports of these various workers and from results obtained from experiments during the past few years that there may possibly be several diseases of the dog included under the name "canine distemper." Opportunity presented itself for comparative studies of naturally infected cases and the experimental virus distemper, when a supply of dried dog virus was kindly furnished the author by Major G. W. Dunkin, National Institute for Medical Research, Mill Hill, England.

In this report are given the results of the injection of this dried virus into a number of animals retained from other experiments, together with suitable controls. These animals included dogs which had recovered from distemper caused by contact with naturally infected cases, and dogs immunized by means of anti-bronchisepticus serum and bacterins and later exposed to naturally infected cases by contact. On account of the similarity of infection of the ferret with the virus of experimental dog-

\*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

distemper to the distemper occurring naturally in these animals and in fitch,\* the results of observations on the disease in these animals, as well as fitch- and ferret-distemper in dogs, also are included in this report.

#### DISTEMPER OF THE DOG BY NATURAL INFECTION

The symptoms of distemper in the dog following natural exposure, while somewhat variable, are characterized by an incubation period of seven to ten days, at which time there is usually an elevation of temperature (103 to 104° F.), accompanied by some malaise, lessened appetite, and roughened hair coat. Usually there is present at this time a serous nasal discharge, elimination of which is augmented by considerable sneezing. Within 24 hours the nasal discharge usually becomes mucous in character and the typical deep cough develops. In these early stages there may be no involvement of the eye, little or no serous discharge being present. In later stages there may or may not be purulent ocular discharges.

The conjunctiva does not become so severely congested as it does following experimental virus infection referred to later in this publication. Diarrhea is not a constant symptom and may not be present during the course of the disease which may last for a period of four to five weeks. In young puppies, however, diarrhea is seen sometimes but no free blood has been noted in the fecal material from the numerous animals observed.

The sick dogs, as a rule, do not emaciate rapidly. In many cases the disease may be complicated by bronchopneumonia and, as a result, the temperature will be seen to fluctuate from day to day. This may continue for three to four weeks, when recovery takes place or it may terminate fatally, depending somewhat on the pneumonic complications.

In advanced cases, there is present a typical odor, difficult to describe, but, when once detected, it is not difficult to recognize in other cases.

In the author's studies usually no difficulty was experienced in the isolation of *B. bronchisepticus* from animals destroyed when the first symptoms were shown. Bacteriological studies of a large number of cases, showing symptoms similar to those described above, indicated that *B. bronchisepticus* was the causative factor in the majority of them. In a few cases streptococci and, in a smaller number, organisms of the colon-typhoid group were present and might have been the etiological factors. In

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\*European polecat.

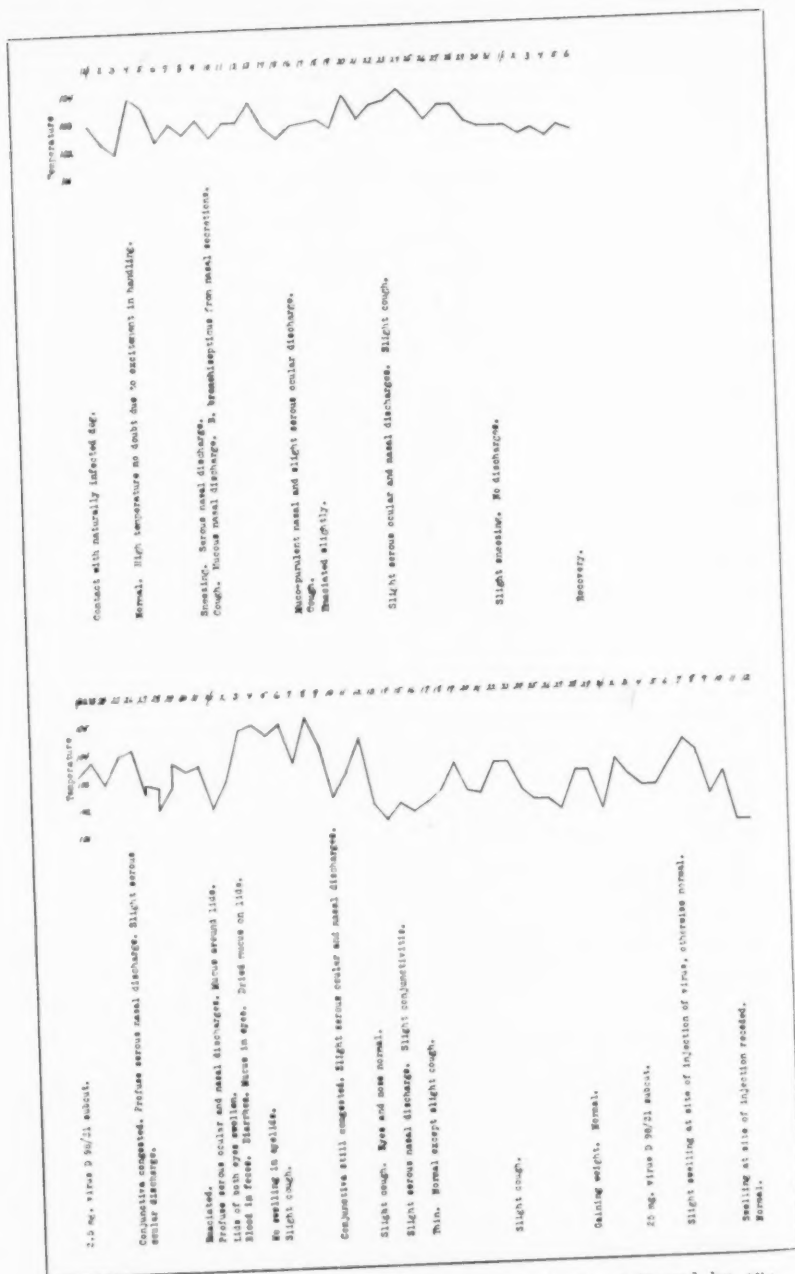


FIG. 1. Clinical charts of dog 42. Natural infection followed by experimental virus distemper.

such naturally infected cases, a filtrable virus was not demonstrated as a causative factor.

As an example of natural infection in the dog, the history of dog 42 is given. This animal was about three months of age at the time of exposure to distemper by contact with a sick dog (fig. 1). Four days after exposure, there was noted a rather marked elevation of temperature, although the dog was otherwise apparently normal. In the experiment for which this dog was being used, it was necessary to take it into the laboratory each day, a distance of about two city blocks, where the temperature was taken after removal to the laboratory. The fourth day after exposure was also the first of the daily trips into the laboratory, the excitement of which no doubt caused some thermal increase. Records of temperatures made later on this and other dogs showed an increase of from 1 to 2° F. after the dogs had been moved.

Regardless of this rise in temperature, the dog remained normal until the ninth day, when sneezing and a serous nasal discharge were noted. One day later, this discharge had become mucous in character and a cough was noted. *B. bronchisepticus* was recovered from the nasal secretions on this day. After eight days of illness, the animal showed practically the same symptoms plus a very slight serous ocular discharge. There was also some slight falling off in condition, but the emaciation was not exceptionally marked. The symptoms described continued with some variation in intensity from day to day, gradually diminishing until recovery took place 37 days after the initial exposure. This dog was held in quarters where distemper was constantly present during its illness and for 16 days after recovery, when it was removed to another building and injected with the virus of experimental dog-distemper (Dunkin and Laidlaw).

Beginning on the fourth day after subcutaneous injection of 3.5 mg. of this virus, there followed a chain of symptoms almost identical with Dunkin and Laidlaw's description<sup>10</sup> of experimental dog-distemper.

There was noted the initial rise in temperature (fig. 1), accompanied by serous ocular and nasal discharges, followed by a fall in temperature, which continued for only a few days. This was followed by the second febrile period, which continued approximately 10 days. During this time the dog had become very much emaciated. The eyelids were swollen and edematous, while the discharges from the eyes were mucous in character. Diarrhea was not severe but considerable free blood was found in the

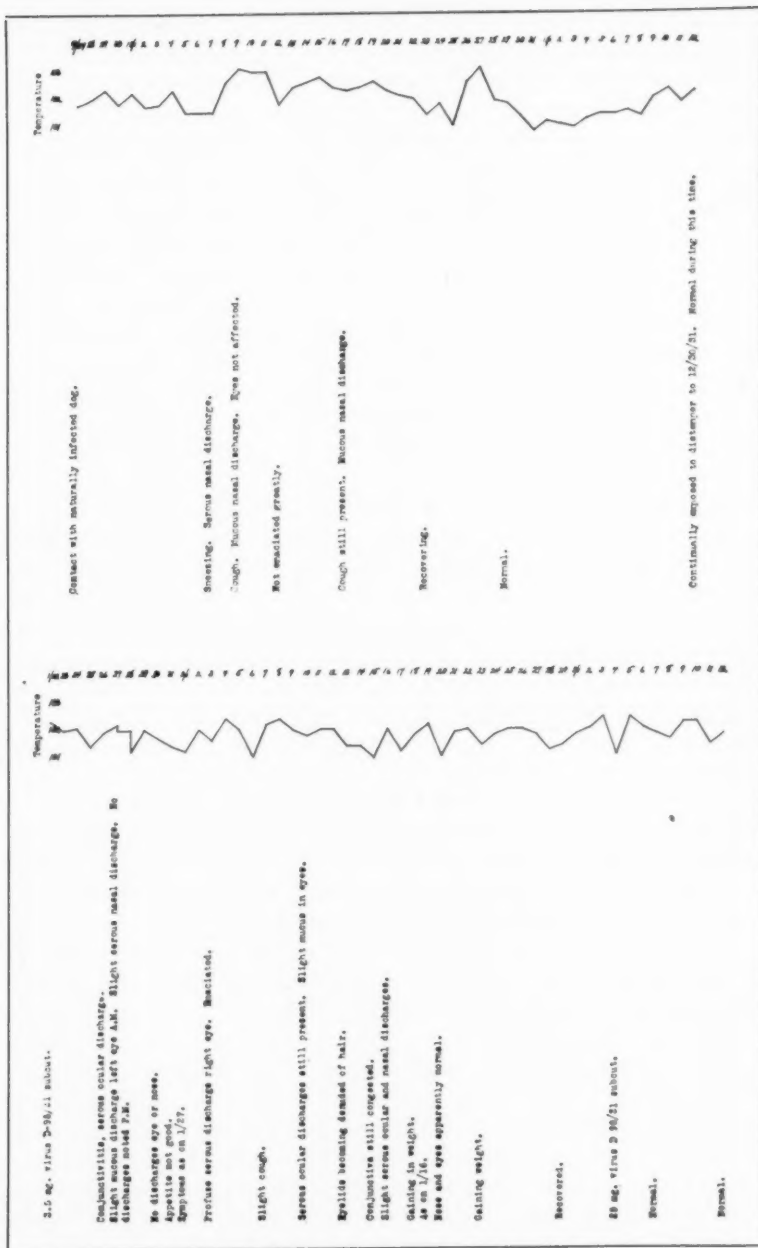


FIG. 2. Clinical charts of dog 37. Natural infection followed by experimental virus distemper.



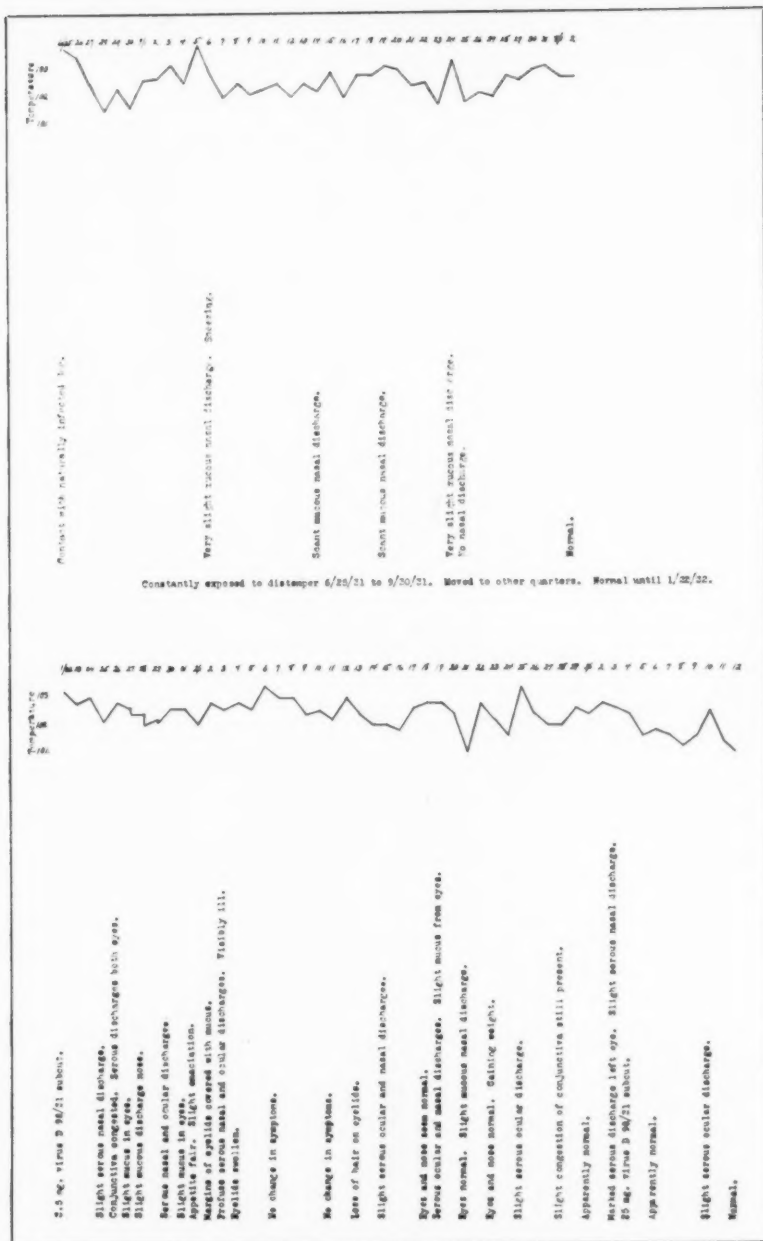


FIG. 4. Clinical charts of dog 10. Natural infection followed by experimental virus distemper.

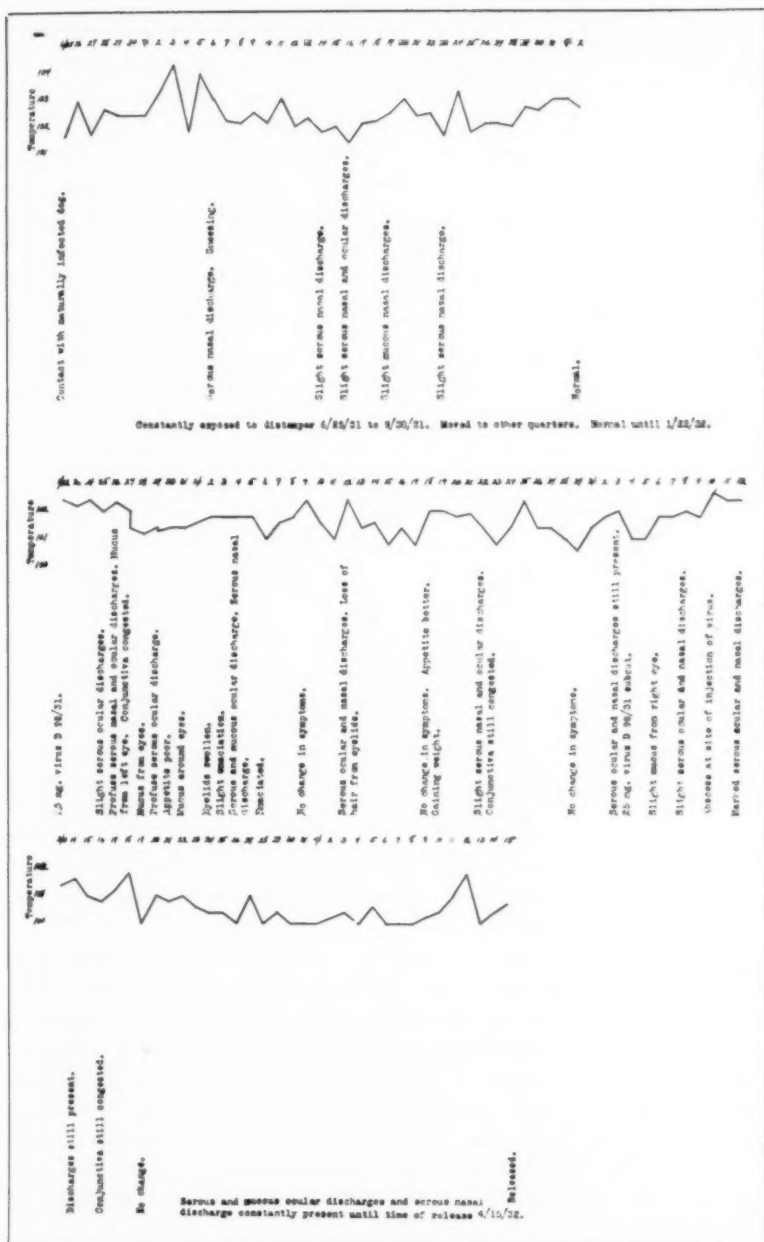


Fig. 5. Clinical charts of dog 11. Natural infection followed by experimental virus distemper.

fecal discharge. The dog seemed well on the road to recovery 26 days after injection of the virus. Forty-two days after the first injection of virus, this dog was given an additional 25 mg. of the same virus without any reaction other than a slight local one and a slight thermal rise on the third day. In both instances these amounts of virus injected into control dogs caused the symptoms described by Dunkin and Laidlaw<sup>10</sup> in the prescribed time (see dogs 47, 48 and 49, figs. 6 and 7, and dog 65, fig. 8).

Another dog (37, fig. 2) which had recovered from natural infection also was given 3.5 mg. of the dried dog virus. The thermal curve described later was not produced but all other typical symptoms developed. Recovery took place after 30 days. This dog also proved to be immune to a massive dose of the virus injected 43 days after the first.

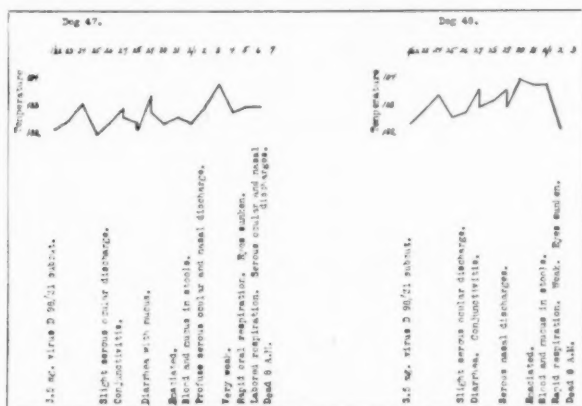


FIG. 6. Clinical charts of dogs 47 and 48. Experimental virus distemper.

Three pups (9, 10 and 11, figs. 3, 4 and 5), raised in quarantine and known to have been free from disease, were treated, while in quarantine, with antibronchisepticus serum and a series of injections of bacterin made from *B. bronchisepticus* cultures. Later they were exposed to distemper by contact with a naturally infected case. These dogs were confined in one cage and were required to eat and drink from the same vessels. After 5 days, the naturally infected dog was removed for autopsy and culturing. *B. bronchisepticus* was recovered from the trachea and lungs.

The three treated dogs developed only very mild cases of distemper even though they were kept in infected quarters for over



FIG. 7. Clinical charts of dog 49. Experimental virus distemper followed by natural infection.

three months. Seven months after the initial exposure to distemper by contact with the naturally infected case, each dog was injected with 3.5 mg. of virus of the experimental distemper.

With the exception of the thermal curve, typical symptoms of this disease were produced, beginning on the fourth day after injection (figs. 3, 4 and 5). Control dogs, described in greater detail later in this publication, under the heading, "Experimental Virus Distemper in Dogs," developed similar symptoms following the injection of the same amount of virus (dogs 47, 48 and 49, figs. 6 and 7).

Forty-two days after the first injection, each of these dogs was given an additional 25 mg. of the virus. Dog 9 showed no reaction whatever but dog 10 reacted very slightly on the sixth day. These dogs were observed for 14 days, during which time no other abnormal symptoms were noted. At the time of the second injection, dog 11 (fig. 5), still showed marked serous ocular and nasal discharges, and, on some days, considerable mucus from the eyes. These conditions were not aggravated by this second injection, but although the temperature remained normal, they were still present when the dog was released 42 days after this second injection of virus.

#### EXPERIMENTAL VIRUS DISTEMPER IN DOGS

The symptoms of this virus disease in dogs, described in detail by Dunkin and Laidlaw,<sup>10</sup> present a different picture than do those which follow natural infection. Briefly the symptoms of the virus disease are as follows: After an incubation period of four days, there is a marked rise in temperature (104 to 105° F.) which may continue for only about 24 hours, followed by a return to normal. Within 48 hours the temperature again increases gradually, to reach greater heights than before. There is usually a severe congestion of the conjunctiva accompanied by a serous discharge from the eyes. Later, considerable mucus is secreted from the eyes and collects around the margins of the lids to form dry or half-dried crusts. Keratitis may develop in the later stages of the disease. The nasal discharge is at first serous and may become mucopurulent later. Diarrhea is a fairly constant symptom, the stools being of a slimy, offensive nature and may contain streaks of bright blood. Due to the lack of appetite and the diarrhea, the animal emaciates very rapidly. In the experience of the author, the disease may terminate fatally within two weeks after exposure or the patient may recover after five to six weeks.

Such symptoms occurred in dogs 47, 48 and 49 (figs. 6 and 7) following the subcutaneous injection of 3.5 mg. of the dried dog virus. These dogs were about five months old and had been raised in quarantine free from any infection. Dogs 47 and 48 showed some slight thermal increase two days after injection of the virus but other symptoms were not manifested until the fourth day. In both cases the disease was very acute, proving fatal to dog 47 in 15 days and to dog 48 in 12 days. Organisms recovered from the internal organs of these dogs were only post-mortem contaminants.

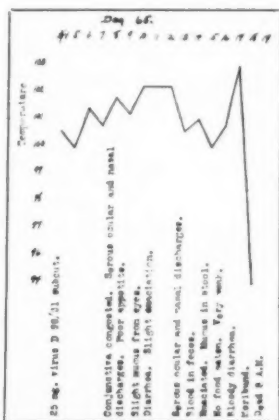


FIG. 8. Clinical chart of dog 65. Experimental virus distemper.

The results in dog 49 were more favorable, since recovery was complete, 38 days after injection of the virus (fig. 7). That immunity to this virus had been established was shown by the fact that no reactions other than a local one and a slight elevation of temperature were produced by injection of 25 mg. of dried dog virus 43 days after the first infecting dose. This amount of virus injected into the control (dog 65, fig. 8) was sufficient to cause the disease which terminated fatally on the fourteenth day.

Twenty-nine days after this massive dose of virus had been given, dog 49 was placed in contact with a naturally infected dog and was permitted to remain so for a period of thirteen days. At this time the dogs were destroyed for autopsy and culturing. *B. bronchisepticus* was recovered from the trachea and lung of the contact dog. Dog 49 developed slight symptoms of distemper

on the seventh day. When destroyed, *B. bronchisepticus* was recovered from the trachea and from the lungs, which showed marked bronchopneumonia.

To determine the effect on normal susceptible dogs when placed in contact with dogs suffering from this virus disease, two 5-month-old dogs (50 and 51, fig. 9), which had been raised in quarantine, were placed in the same cage with controls 47, 48 and 49. Contact first was made six days after the control dogs had been injected with the virus. After four days, dogs 50 and 51 showed beginning symptoms of the disease. The infection in both progressed rapidly and terminated fatally on the 23rd and 28th days.

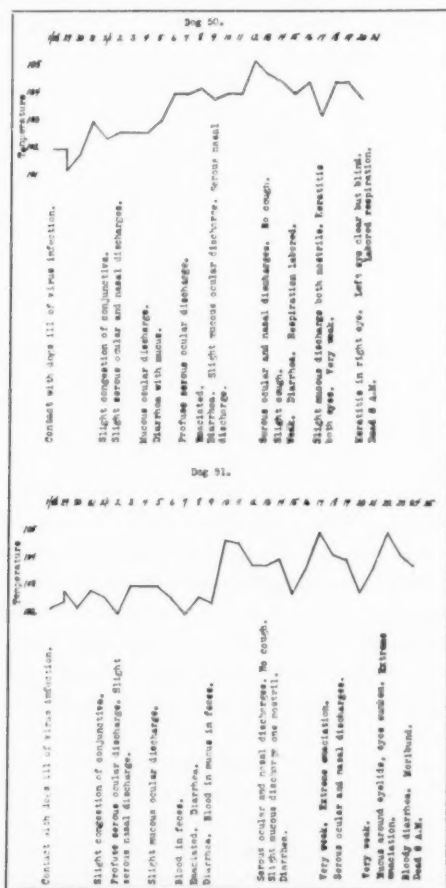


FIG. 9. Clinical charts of dogs 50 and 51.  
Experimental virus distemper  
by contact.

No definite bacteriology of the internal organs was seen after cultures had been made.

#### DISTEMPER IN THE FERRET

Dunkin and Laidlaw<sup>13</sup> describe dog-distemper in the ferret as an "acute infectious fever characterized by an incubation period of about ten days, a coryza at the onset of the illness, and the formation of vesicles and pustules around the mouth." According to these authors the mortality rate is high.

An outbreak of a disease, in which the symptoms shown were similar to those described by Dunkin and Laidlaw<sup>13</sup> as dog-distemper in the ferret, was observed in a group of three ferrets which were being held in quarantine. These animals had been apparently normal during the three weeks they had been isolated. The source of the infection was not determined.

When the first symptoms of disease were noticed in these three ferrets, they were taken into the laboratory for autopsy and study. While the postmortem examinations were being made, the spleens were removed with aseptic precautions and a part of each made into a 20 per cent suspension in sterile physiological salt solution and filtered. These filtrates were then stored in sealed bottles at 2 to 5° C. for 17 days, when ferrets and pups were available for test purposes.

The remainder of each spleen was minced finely and dried in vacuum over  $P_2O_5$ . When drying was almost complete, the spleen tissue was ground finely, dried again and stored at 2 to 5° C. in vacuum over  $P_2O_5$ . These samples were held for 17 days, when ferrets and pups were injected subcutaneously with 50 mg. and 200 mg., respectively.

Two hundred mg. of dried spleen from the first ferret (D-F-1), injected subcutaneously into a dog, caused general symptoms of the experimental dog-distemper described by Dunkin and Laidlaw<sup>10</sup> within four days. A typical thermal curve was not obtained. This dog died on the eighth day after injection. A litter-mate of this pup, which was injected with 2 cc of sterile filtrate of spleen suspension, died in six days without manifestation of the typical symptoms. Two ferrets, one injected with 50 mg. of dried spleen, the other with 2 cc of the filtered suspension, remained normal for 32 and 30 days following injection. Both contracted fitch-distemper, which also was being studied at this time, and described later in this report. These ferrets died 14 and 10 days, respectively, after development of the first symptoms.

Pups injected with dried spleen and filtrates of spleen suspensions from the second ferret (D-F-2) sickened four days after injection. The first died after 18 days, while the second lived three days longer. As an example, the record of dog 28 (fig. 10) is given. All symptoms in these pups except the thermal curves were typical of experimental virus distemper in the dog described by Dunkin and Laidlaw.<sup>10</sup>

Ferrets, injected with dried spleen and filtrates of spleen suspensions, remained normal for 30 and 38 days. Both contracted the fitch-distemper, referred to above, but recovered in 30 days and one week respectively. Both these animals were found to be immune to the experimental dog-distemper when injected seven

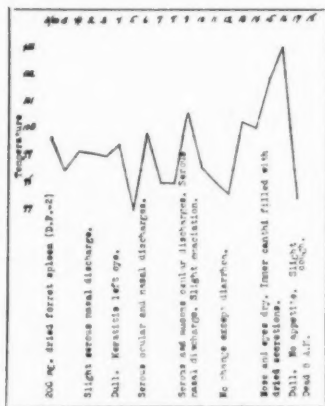


FIG. 10. Clinical chart of dog 28. Ferret-distemper in the dog.

months later with 3.5 mg. of dried dog spleen received from Major Dunkin. This amount was sufficient to produce symptoms of the disease in a control ferret in nine days, followed by death four days later.

Spleen tissue and filtrate from the third ferret (D-F-3) produced typical symptoms in two pups injected, with the exception of the thermal curve. The one which had received the dried spleen died 16 days after injection while the other died in 11 days.

These materials injected into ferrets caused no reaction in 36 and 33 days. These animals, too, contracted the fitch-distemper, but recovered after 30 and 23 days of illness. Seven months later, both resisted infection with 3.5 mg. of the dried dog spleen.

Cultures from all the animals dead after injection of the tissues or filtrates from ferrets were negative.

#### DISTEMPER IN THE FITCH

During the spring of 1931, an opportunity was presented to study distemper in the fitch, symptoms of which recently have been described in detail by Dalling.<sup>14</sup> There was brought to the attention of the author a sick animal which was one of a group of twelve which had been imported from England about six weeks before the outbreak occurred. Inquiry of the owner revealed that no dogs were kept on the premises nor did the attendant have any contact with any other animals. A definite source of infection was not determined.

The owner stated that six out of the twelve animals already had died, the symptoms shown having been similar to those shown by the one presented for study. These were comparable to those described by Dalling, as well as to the symptoms of dog-distemper in the ferret described by Dunkin and Laidlaw.<sup>13</sup>

This one animal (F-1) was destroyed by intramuscular injection of strychnin sulfates and a careful autopsy performed. During this postmortem examination, the spleen was removed carefully, ground finely and made into a 20 per cent suspension. This suspension was held at 2 to 5° C. for 48 hours, when 2 cc were injected into a normal ferret (F-F-1).

Ten days later, this ferret showed evidence of infection, manifested by symptoms similar to those described by Dalling as occurring in distemper in the fitch. The condition of this ferret gradually grew worse until death took place on the 26th day after injection.

In order to determine the infectious nature of this disease of the fitch by contact, a normal ferret (F-C-F) was placed in the cage with the ferret (F-F-1) four days before it succumbed to the disease. On the tenth day, this ferret (F-C-F) seemed sluggish, and the tops of the feet had become quite red. The next day the eyes were half-closed and there was present some discharge. A slight nasal discharge also was seen. The ocular secretions gradually became mucopurulent in character, and at this time there was noted an intense reddening of the lower lip. Death took place on the 27th day after contact with the infected ferret F-F-1.

Further evidence of the infectious nature of the disease was shown by the results obtained after a normal ferret had been placed in a separate cage in the quarters where these ferrets

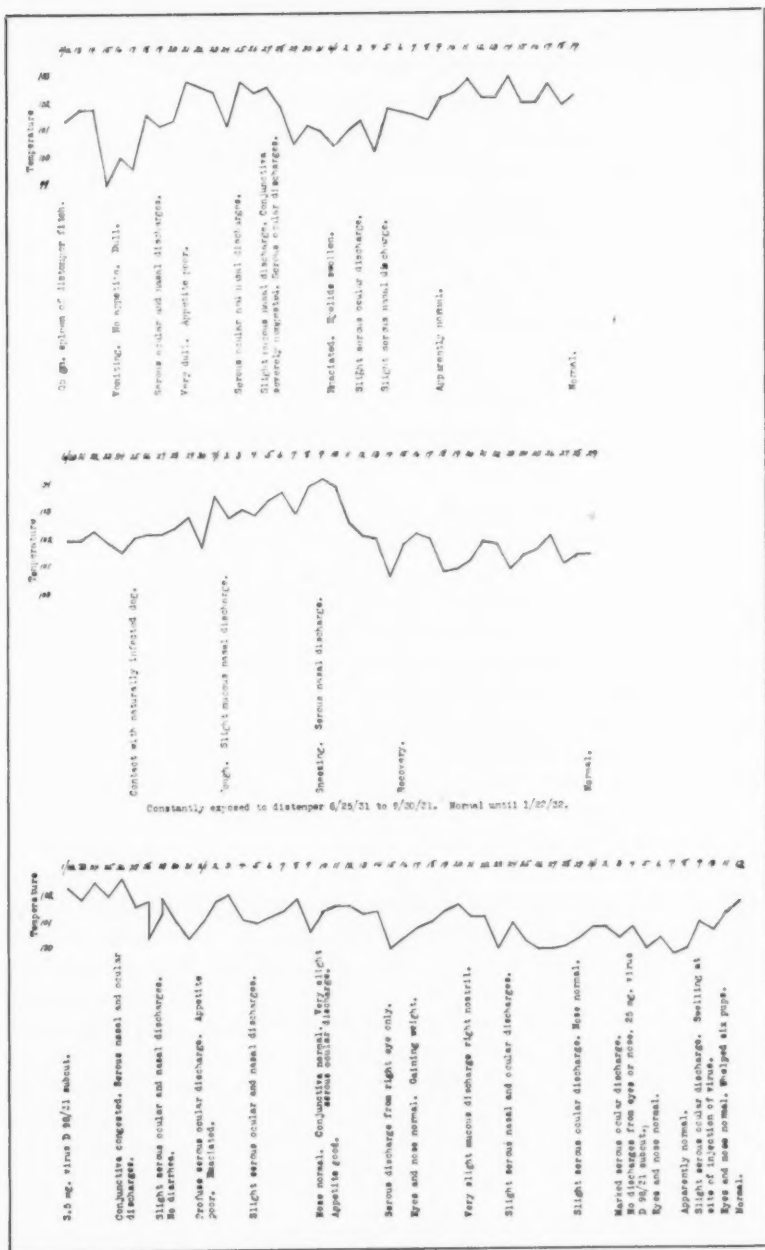


FIG. 11. Clinical charts of dog 7. Fitch-distemper in the dog followed by natural and experimental virus infections.

were kept. No particular effort was made to infect this animal, but evidently the infection was carried to it by the attendant, since symptoms developed on the tenth day. Symptoms shown during the course of the disease, which terminated fatally on the 22nd day after being placed in the quarters, were similar to those seen in F-F-1.

At this time a number of ferrets, which had been injected with filtered and unfiltered spleen suspensions of the ferrets suffering from distemper, were being held in these same quarters for observation. A ferret which had been injected with unfiltered suspension of spleen from a naturally infected dog also was held in these quarters. The disease of the fitch which had been transmitted to several ferrets (F-C-F and control) spread through the other animals, with the result that all were affected. Recovery in these animals took place in from 14 to 35 days.

In order to determine that the danger of infection to other animals had passed after recovery of these ferrets, another normal ferret (control 7-17) was placed in these quarters and observed. No evidence of infection was seen for seven months.

These ferrets were held under observation for a period of approximately seven months, when each was injected with 3.5 mg. of dried dog-distemper virus. All proved to be immune except control 7-17, which showed evidence of infection on the ninth day and died four days later. Two normal ferrets, held in quarantine for three months prior to injection with 2 and 4 mg. each of this same virus, sickened on the tenth day and died 13 and 14 days later.

The infectiousness of fitch-distemper for dogs was shown by the results obtained following the injection of 0.5 mg. of spleen from fitch F-1 into dogs 7 and 8 (figs. 11 and 12). Both these dogs developed symptoms of the experimental dog-distemper described by Dunkin and Laidlaw.<sup>10</sup> After recovery both dogs were exposed to distemper by contact with a naturally infected case. Both developed typical symptoms. Dog 8 was destroyed twelve days after contact with the naturally infected case. *B. bronchisepticus* was recovered from the lung.

Dog 7 recovered from natural distemper and was kept in infected quarters for over three months. No further evidence of disease was noted. Four months later, this animal was injected with 3.5 mg. of dried dog virus and, while the thermal curve was not typical of experimental dog-distemper, other symptoms shown led to the conclusion that the dog was not immune (fig. 11). This dog later proved immune to a massive dose of this same virus.

Another opportunity was presented during the early part of 1932 to study distemper in the fitch, when one of these affected animals was presented for examination. This fitch was destroyed and the spleen removed for injection into dogs. Dog 53 (fig. 13) was injected with 1 gm. of this spleen tissue and developed the thermal curve and other symptoms typical of Dunkin and Laidlaw's experimental dog-distemper. Death took place on the 23rd day after infection. Cultures from the internal organs were not definite.

Dog 52 (fig. 14) was injected with 0.5 gm. of fitch spleen and recovered from an attack of a disease similar to experimental dog-distemper after 33 days. This dog later was injected with 25 mg. of dried dog-distemper virus. Evidently the immunity

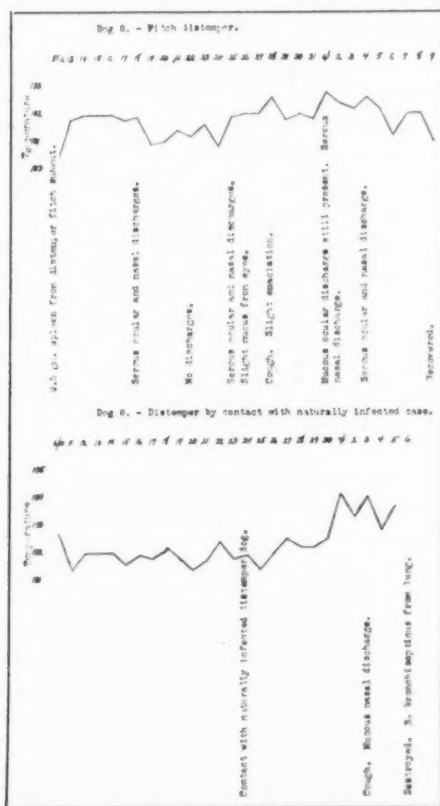


FIG. 12. Clinical charts of dog 8. Fitch-distemper in the dog followed by natural infection.

to dog-distemper virus was of low order following recovery from fitch-distemper, since there was a slight rise of temperature on the third day, followed by a marked keratitis and edema of the eyelids a few days later. However, this reaction may have been due partly to the weakened condition of the animal at the time of injection. Recovery except for extreme emaciation was complete after two weeks. This dog was exposed later to distemper by contact with a naturally infected dog and developed symptoms of infection. When an autopsy was made after nine days of illness, *B. bronchisepticus* was recovered in pure culture from the trachea, lungs and heart-blood. Likewise, this same organism was recovered from the same organs of the dog used for contact.

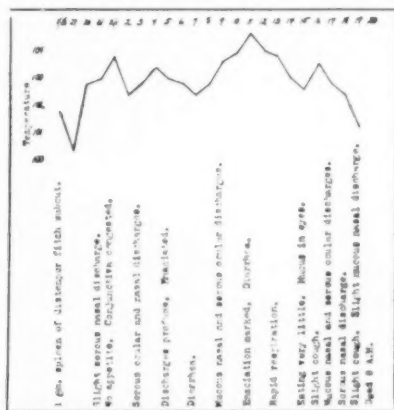


FIG. 13. Clinical chart of dog 53.  
Fitch-distemper in the dog.

### DISCUSSION

Anyone who has had contact with a considerable number of cases of dog-distemper will concede that the symptoms are variable, depending somewhat on the age and condition of the animal at the time of exposure as well as on the virulence of the infection.

It has been the author's observation that no difficulty is experienced in producing the typical symptoms of natural infection, described earlier in this report, when susceptible pups are placed in contact with naturally infected cases, the majority of which have been found to be caused by *B. bronchisepticus*. These symptoms are different from those described by Dunkin and Laidlaw<sup>10</sup> as caused by the filtrable virus with which they were working.

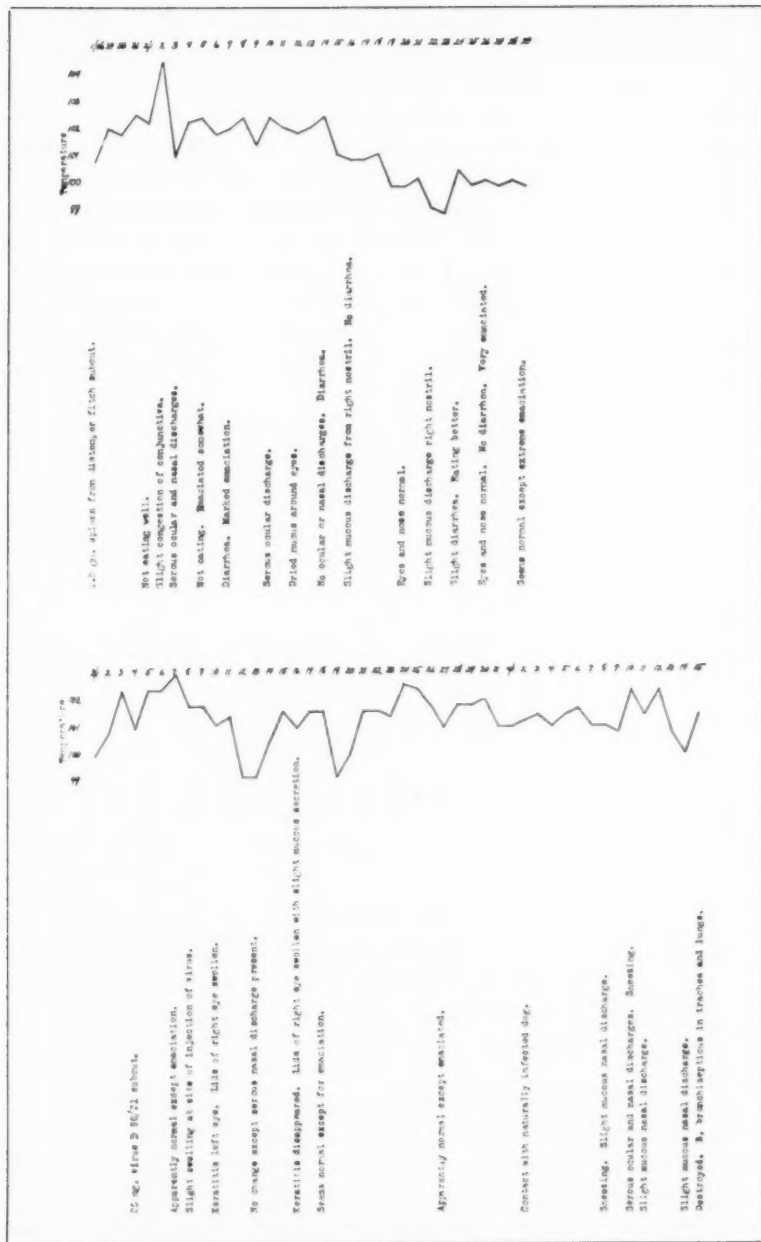


FIG. 14. Clinical charts of dog 52. Fitch-distemper in the dog followed by experimental virus and natural infections.

Since it is generally conceded that one attack of distemper results in immunity to further infection, the results of the injection of filtrable virus into dogs which had recovered from natural infection were observed with considerable interest.

The results obtained in dogs 42 and 37 (figs. 1 and 2) indicated that natural infection and the filtrable-virus disease were separate and distinct and that the immunity which resulted from the first did not protect against the latter.

Further evidence that this was true is shown by the results obtained in dogs 9, 10 and 11 (figs. 3, 4 and 5). These three dogs had been immunized, while in quarantine, with antibronchisepticus serum and bacterin and later exposed to infection by contact with a naturally infected case. Although this immunity was not 100 per cent, since each animal developed a very slight attack of distemper, it must be remembered that the test was exceptionally severe, in that these dogs ate, drank and slept with a typical case of the disease for more than five days. This is considerably more exposure than would occur under ordinary conditions.

All these dogs later proved susceptible to a small dose of the virus, developing practically all the symptoms of this disease with the exception of the thermal curve. All proved to be immune to a massive dose of this virus given later.

Prior to injection of this filtrable virus, these five dogs were kept in infected quarters for varying periods up to three months. Since there was one or more cases of distemper always present and since new dogs had been received weekly from all parts of the city, it would seem that, during the time the five dogs were held with these cases, if a filtrable-virus disease has been present, these dogs, being susceptible, would have acquired the disease and later proved resistant to infection following injection of the virus. Since this was not the case, the results tend to prove the conclusions of former experiments, in that these naturally infected cases were of bacterial origin (*B. bronchisepticus* in a majority of the cases) and a filtrable virus played no part in producing the symptoms.

Still further evidence that the two conditions are separate and distinct is shown by the results obtained in dog 49 (fig. 7). This dog recovered from the filtrable-virus disease following the injection of 3.5 mg. of the virus, but notwithstanding the fact that it later proved to be immune to further infection with this

virus, it was susceptible to infection following contact with a naturally infected dog.

Dogs raised in quarantine proved to be highly susceptible to virus infection, whether by subcutaneous injection or by contact. The mortality in these cases was extremely high (83.3 per cent). The disease produced in these dogs was comparable to that described by Dunkin and Laidlaw.<sup>10</sup> Bacteriological studies of the internal organs of these dogs dead of virus infection were indefinite.

Injection of susceptible pups with filtered and unfiltered suspensions of spleens from ferrets suffering from distemper resulted in symptoms of a disease comparable to that produced by the experimental dog-distemper virus. Unfortunately all the dogs injected died, so that tests to determine the immunity to natural infection could not be made. The fact that the ferrets, injected with these materials from the diseased ferrets, failed to develop symptoms of the disease may have been due to extreme resistance on the part of the test animals. However, these animals later did become infected with the distemper of the fitch, which proved fatal to two out of the six used.

A study of an outbreak of distemper in the fitch indicated that this disease was similar to that produced in ferrets by the virus of dog-distemper and also similar to distemper occurring naturally in the ferret. Ferrets that recovered from distemper contracted from the fitch later proved to be immune to the experimental dog-distemper virus.

Dogs injected with suspensions of spleens from these diseased fitch developed symptoms similar to those following injection with the experimental dog-distemper virus. This infection did not lead to immunity against natural infection as shown by dog 7 (fig. 11). Evidently the immunity produced in this dog by the injection of tissue from the fitch was not of so great duration as occurred in ferrets, since with the exception of the thermal reaction, this dog developed symptoms of the virus disease after injection of 3.5 mg. of the experimental dog-distemper virus.

Another dog (52, fig. 14) showed considerable reaction to the effect of the dog-distemper virus after recovery from the infection of the fitch. This may have been due partly to the massive dose given and to the fact that this dog was still in a very weakened condition at the time the experimental dog-distemper virus was injected. Later tests showed that this animal was susceptible to distemper by natural infection.

## SUMMARY

Symptoms of distemper occurring in dogs following exposure to naturally infected cases are described. Recovery from such infection did not result in immunity, in the dogs used, to the virus of experimental dog-distemper.

On the other hand, recovery from the filtrable virus disease did not result in immunity to the natural infection, the majority of which cases are caused by *Bacillus bronchisepticus*.

Dogs raised in quarantine were found to be very susceptible to both natural infection and the filtrable virus. The majority rate in this latter disease was exceptionally high (83.3 per cent of the dogs used).

A study of an outbreak of distemper in ferrets indicated that this disease was similar to, if not identical with, the disease of these animals produced by the virus of experimental dog-distemper. Symptoms produced in dogs following the injection of filtered and unfiltered suspensions of spleens from the affected ferrets were similar to those produced in dogs by the virus of experimental dog-distemper.

The distemper of the ferret was not transmitted to other ferrets by injection of filtered or unfiltered suspensions of the spleens taken from the affected animals. This may have been due to extreme resistance of the test animals.

These same ferrets later were found to be susceptible to the distemper of the fitch, a study of which showed that it was similar to the disease in ferrets, and similar to the disease in ferrets caused by the virus of experimental dog-distemper.

The symptoms of fitch-distemper in the dog were seen to be similar to those caused by the virus of experimental dog-distemper. The dogs recovered from fitch-distemper developed an immunity to the virus of experimental dog-distemper which was of low order. Recovery from this disease did not result in immunity to natural infection.

## CONCLUSIONS

A comparison of the symptoms seen occurring after natural infection with those produced in dogs by the injection of the filtrable virus indicate that these two diseases, both of which are called canine distemper, are separate and distinct.

This conclusion is substantiated further by the fact that the dogs which had recovered from natural infection, the majority of which cases have been found to be due to *Bacillus bronchisepticus*, were not immune to the virus of experimental dog-

distemper, and, *vice versa*, dogs recovered from infection with this virus were not immune to natural infection.

Comparison of the symptoms of ferret-distemper in the ferret, fitch-distemper in the ferret and infection with the virus of experimental dog-distemper in these animals leads to the conclusion that these diseases are similar.

Ferrets recovered from fitch-distemper possessed a solid immunity against infection with the virus of experimental dog-distemper but dogs recovered from fitch-distemper developed either an immunity of low grade or of short duration to infection with this virus.

Symptoms of ferret-distemper, fitch-distemper, and the infection with the virus of experimental dog-distemper in the dog were found to be similar to but different from those seen following infection by contact with naturally infected cases.

Dogs recovered from fitch-distemper were not immune to infection by contact with naturally infected cases.

From the limited number of animals used in these experiments it would seem that while fitch-distemper, ferret-distemper and infection with the virus of experimental dog-distemper in the dog are similar, if not identical, these conditions are entirely different from the disease in dogs as it occurs following contact with naturally infected cases, the majority of which have been found to be caused by *B. bronchisepticus*.

#### ACKNOWLEDGMENT

Acknowledgment hereby is made to Major G. W. Dunkin, National Institute for Medical Research, Farm Laboratories, Mill Hill, England, for his kindness in supplying a quality of dried dog-distemper virus by which a part of these comparative studies were made possible.

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### Committee on Local Arrangements

The Committee on Local Arrangements for the seventieth annual convention of the A. V. M. A., scheduled for Chicago, August 14-18, 1933, has been organized as follows:

*Chairman:* J. V. Lacroix, 1817 Church St., Evanston.

*Secretary-Treasurer:* H. Preston Hoskins, 1230 W. Washington Blvd., Chicago.

#### SUBCOMMITTEES

*Alumni Meetings:* C. N. Bramer, 1817 Church St., Evanston.

*Army:* Maj. F. B. Steinkolk, 1819 W. Pershing Rd., Chicago.

*B. A. I.:* G. E. Totten, 999 Exchange Ave., Chicago.

*Banquet and Reception:* R. L. Tinkham, 112 W. Division St., Chicago.

*Clinics:* L. A. Merillat, 1827 S. Wabash Ave., Chicago.

*Entertainment:* C. L. Miller, 240 Madison St., Oak Park.

*Exhibits:* E. E. Sweebe, c/o Abbott Laboratories, North Chicago.

*Finances:* D. M. Campbell, 75 E. Wacker Drive, Chicago.

*Hotels:* H. Preston Hoskins, 1230 W. Washington Blvd., Chicago.

*Parking Stations:* L. T. Kilfoy, 2508 Lawrence Ave., Chicago.

*Press and Publicity:* J. V. Lacroix, 1817 Church St., Evanston.

*Registration:* E. L. Quitman, 1514 W. Van Buren St., Chicago.

*Transportation:* M. A. Sweeney, 6523 South Park Ave., Chicago.

Meetings of the Committee are being held on the first Thursday of each month, at the Palmer House.

### What to Use for Money

If the farmers are unable to pay cash (35 cents per horse) for bot control treatment in Grundy county, Illinois, the veterinarians there have agreed to take grain, poultry or eggs as payment. Grundy is one of sixty counties in the state where bot-control projects are in progress, according to Dr. Robert Graham, of the University of Illinois.

### Cooperation

The Mississippi State Veterinary Medical Association has an agreement with the Mississippi State Board of Health, providing that the latter body will not employ any veterinarian who is not an active member of his state association.

## THE HOSPITALIZATION AND CARE OF CATS\*

*By H. W. BROWN, Fort Wayne, Ind.*

The hospitalization and care of cats presents to the veterinarian quite a different problem from that of hospitalizing and caring for dogs. In some instances, this can best be shown by comparison, one with the other. The feline population of the United States today is far greater than ever before and increasing every year. There is also a marked improvement in the breeds, and a greater demand by cat fanciers for pure-bred cats.

In my experience as a veterinarian, I have never come in contact with any group of fanciers that are as exacting, or that are as devoted to their pets as the cat fanciers, nor have I contacted any group of pure-bred breeders, who have a better knowledge of scientific breeding to obtain the desired results, even to color, which is not practiced in any other animals where there is such a variety of colors.

As a result of these facts, veterinarians will be called on more often to treat these animals as time goes on, and it behooves every one of us to have or acquire a working knowledge of them, in order that we may talk intelligently to their owners.

I do not pretend to be an authority on the subject assigned me for this paper, but the care and hospitalization of cats was thrust upon me, as has been true of every veterinarian in city practice, and I found it imperative to have in my equipment a working knowledge of the breeds, or refuse the service. I chose the former.

Where one can acquire this information is the first question to arise. There are a number of books published on diseases, care and management of the cat, most of which have been written by fanciers in England, and much can be learned as regards management from these books. Another source of information is to be found in the journals published monthly by the national cat clubs. There are only a few books written by veterinarians, and these pertain more to the diseases of cats.

If we are to be successful in treating sick animals we must first have an intimate knowledge of them when well, and with no animal which we are called on to treat does it mean quite so much to be familiar with their habits as it does with cats. I know of no better method to get to know the cat better than to raise a few and, if possible, make a pet and companion of one or two of them. I assure you that you will get some first-hand information that can be obtained in no other way.

\*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

I do not mean that we should go into a wholesale breeding game and raise cats to sell, for we would then be competing with our own clients, which creates an undesirable situation, but if only one or two litters are raised and given away, we have gained some first-hand information on the care and handling of cats, and at the same time instilled a confidence in a number of fanciers, that you like cats and are really interested in them.

We have in the United States today two common breeds of cats, namely, the short-haired and the long-haired or Persian. The long-haired cats often are incorrectly called Angora, for the Angora is considered to be only a strain of the Persian, and today all long-haired cats are called Persians and so registered by the cat clubs of America.

It is the opinion of many, that Persian cats are more delicate than the short-haired varieties, but, as far as the veterinarian is concerned, they are the same as regards their care and treatment. In handling cats the first essential to obtain best results is to learn their restraint, keeping in mind at all times that you are restraining a domesticated wild animal that may at any moment, through fright, revert to its wild traits and use every method of attack in its power to gain its freedom.

#### HANDLING AND RESTRAINT

To handle cats with safety to yourself, and still gently enough to gain the approval of the owner standing by, makes it a very difficult task. In fact, many of them could be handled with tongs, as we do foxes, much more easily if the owner would permit, but any rough handling of a cat by a veterinarian, regardless of how vicious and wild the animal may be, if tolerated by the owner, is not appreciated and will in the end mean the loss of a client, or his confidence in your ability to handle cats successfully.

When handling cats you need not only to protect yourself from bites, but also beware of their claws, for they can do even more damage with any one of their four feet. Considering these facts, the first routine procedure should be to cut the toe-nails as short as possible without injuring the cat, and thus avoid a bad scratch later on in the course of the examination, should one foot get free.

I will not attempt to give in detail the different methods used in the restraint of cats, but will mention a few things which have aided us in their handling.

First, it is not good policy to depend on the owner of the

animal to hold it while you are making the examination, for if the cat becomes excited and starts struggling to get loose, the owner immediately will release all holds for fear of hurting the cat or getting hurt himself, and you are left with only a partial hold. If you are not very quick, you will either be scratched or bitten before you can turn the cat loose, for, as you may have noted, the expression "quick as a cat" did not originate from watching them in slow-motion pictures.

The cat is one animal that, in handling, sometimes requires as much or more skill in releasing a hold on it as it does to get it. It is advisable to have your assistant trained not to release a hold until, by a given signal, both parties release their holds.

#### TAKING THE TEMPERATURE

When a cat is brought to the hospital for examination, every precaution should be exercised to prevent it from becoming excited. A metal or glass-top table is best on which to place them for the examination, as it will prevent them from getting a foothold.

Taking the temperature of most animals is a simple and easy task, and requires no specific technic, but with the cat proper technic is very necessary and important. The thermometer should be well oiled, and at first inserted just beyond the bulb and held in that position until the sphincter muscle of the anus relaxes, which usually is only a few seconds. Then, by gentle pressure and with a rotary motion of the thermometer, insert it the proper distance. This can be accomplished with little or no resistance, but if you try to force the thermometer at first, you meet with a resistance so great that it would make one think there might be an obstruction, and if continued force be used before the anus is relaxed, the thermometer may break and puncture the bowel. Even if that does not happen, forced pressure will cause the most docile cat to become excited and scared so badly that it will require quite some time to get it quieted so you can proceed with the examination.

Cats, unlike dogs, become attached to places, and not to people, and for this reason every effort should be made to ascertain from their owners just how they are treated at home and then try and make their surroundings in the hospital conform to that of their home life as near as it is possible, for the first few days at least. It is not advisable to make too radical a change either in their diet or their surroundings (even though both

are wrong) until they have become accustomed to their new surroundings in the hospital. Inquiry should be made as to their diet at home, and for the first few days the same food provided, gradually changing to the correct diet if they have not been fed correctly.

As stated before, cats become attached to places and not to people, and any radical change either in their diet or surroundings will cause them to become homesick, and they refuse either food or water and continue to do so until they die from homesickness and starvation.

#### GROOMING IS IMPORTANT

Taking care of the coat or proper grooming, especially in Persian cats, is a procedure which I wish to emphasize to you as an important routine procedure in every small-animal hospital. It should be a practice to comb out the coat of every cat in the hospital every morning, and if the coat has become soiled during the night, clean the coat with either ether or rubbing alcohol. Cats abhor anything on their coats, and often cats brought to the hospital have been given a dose of castor oil, or, I should say, a half-dose and the other half smeared all over them trying to get them to swallow it, and in many cases of this sort all that is necessary to restore that cat to normal is to comb out the coat and remove all oil and odor from the coat. The oil the owner gave will produce the needed laxative and the cat will take a new lease on life and proceed to keep the coat clean.

It has been proven to me many times that a simple gastritis may be induced by the soiling of a cat's coat with medicine while trying to administer it and then not removing it. The cat licks the coat and develops a simple gastritis, later on involving the small intestines, and the cat dies within seventy-two hours from gastroenteritis.

One other thing I want to mention in caring for cats. Cats have a very rough tongue which they use as a comb in keeping their coats clean and free from mats or tangles and for this reason it is not advisable to bathe them oftener than is absolutely necessary, for if they are bathed too often they will quit washing themselves and in a very short time the coat becomes ragged, matted and full of dirt and it is not long until you have a sick cat.

Persian cats may be cleaned very nicely by dusting corn meal through the coat and then combing it out, thus avoiding bathing them too often.

In conclusion—to be successful in caring for cats is to know their restraints, and to make their surroundings conform to that of their natural environment or as near as it is possible to do so.

#### DISCUSSION

DR. E. J. FRICK: Mr. Chairman, there is a point I want to mention to this group because it helped me. I notice the author did not mention it. He may know of it, and you may know of it and may practice it. It is familiar to me, and I want to pass it on to this group.

When you go home, go to your shoemaker and have him get a piece of soft leather or chamois. Tell him you want to make a little boxing-glove. Get four of them; take a piece of string and pull these over the cat's feet. Pull them tightly and have soft pieces of leather on the cat's feet. Then you will have to watch only the cat's head, you will not have to be bothered with the claws, and will have a method of handling cats that can not be beat. I am tired of being scratched by cats that I thought would not scratch me, and I appreciate having boxing-gloves handed to me when we have to handle cats.

DR. H. K. MILLER: I am reminded of what Dr. Denner said when I was a student of his in dentistry, floating horses' teeth. He said the best method of holding a horse's head when you float his teeth is to have a negro who goes to sleep. I think the trouble that we have had a great many times with cats is that we hold them too hard. We try to restrain them too much. The less hold we take and the fewer people we have around—the better it is for all. I think Dr. Frick's idea is good. I generally trim their nails and muzzle them, especially if I have a delivery to make or any wound to dress. I generally muzzle the cat for the safety of the owner. If we have one person around, that is sufficient.

There is another thing I have been doing a great deal and that is handling the cat under cover, especially in a house. Put a blanket or a heavy bath towel over the cat to take the temperature, for instance. Play around with the animal for a while and get better acquainted with it. There is one more thing in hospitalization that brings up quite a problem. We find a great many of our cats coming in to board and getting into a strange environment where they will not eat. When you get these cases in and keep them a while, they will sit in the corner and not eat. The first thing you know they have gastritis. In the beginning, we had trouble with the cats we spayed. They would recover from the operation, would go home and come down with gastritis a few days later. We have overcome this trouble by keeping their bowels moving. If we get a cat and find he does not use his pan, we give it either an enema or a 5-grain chocolate-coated cascara pill. If a cat's bowels do not move on the day on which it is spayed, it gets a physic. In that way we have worked on our boarders. When a cat is taken away to be bred and has been carried to some place where it stays two or three days, in a day or two it may get gastritis. Probably you have had that experience. The cat goes over and stays for a couple of days crouched in the corner. Her bowels do not move, and she gets sick. The most important thing in the hospitalization of cats is to see that their bowels move regularly.

As was brought out in the paper, the cat is attached to the home and not to the owner. I recall a case, maybe ten or fifteen years ago, where we had a cat in our country place to board for about four months, and it kept in perfect health. In the meantime the owner changed her home, went to a new one, wanted the cat back, and took

it. Later she telephoned and said her cat was not eating; it looked healthy but would not eat. She called me up every day for about a week—the cat did not eat. Finally she said, "You will have to come up." I said, "All right, I will be up." In a few minutes she called again and said, "Never mind, you need not come, the cat has started eating all right." That is the experience of almost everybody. Get a cat to a place to board and it will never eat the first day. When a cat goes into a new home, it is the same.

DR. FRICK: I have another suggestion which I have found works. I first found it out in puppies. We sell somebody a puppy and he is taken away. The next day the people come in and complain that "this puppy kept us awake all night by howling." I said, "Well, all you have to do is take an old alarm clock and set it right by the side of the puppy's basket and he will go right to sleep." He is used to the ticking of the alarm clock and thinks he has company. Put an alarm clock in a cat's cage, and the animal will become nervous, but the ticking of the clock seems to soothe the dog. Cat psychology is entirely different from dog psychology.

DR. J. V. LACROIX: Having been so fortunate as to have cats for playmates when I was young, I have been a cat's friend for some time. I realize that most veterinarians detest cats and do not like to handle them, and opinions concerning cats in general would not sound very good to the owners of cats and cat fanciers. I think that Dr. Miller and Dr. Frick have both given good advice. I think Dr. Miller's advice concerning the restraint of cats applies also to some breeds of dogs. I think it is well to take the cat in your hands, go to a quiet place, sit down and wait a while, and gain the cat's confidence. When it realizes you are not trying to hurt it, you may do many things without exciting the cat and without getting scratched.

Cats coming in to be hospitalized and boarded for a time certainly in many instances do not like the change in environment. They show resentment by refusing to eat, and if they remain crouched in the cage at night, very much coprostatic results. We practice using a little vaseline which we have put up in a half-ounce tube with a long nozzle, and we give them a little of it. In twenty-four hours, or after three days at the most, we give enemas.

The giving of enemas to a cat may not sound very rational to some of you, but if you will let the cat stand on the drain-board of a sink, and lubricate the end of the nozzle of the syringe with a little soap, this may be done without disturbing the cat. Put the cat on the incline of the board and you will not get scratched. Generally you will not be hurt at all, if you are alive and awake.

In the major operations and in some examinations the manner of handling cats has been stressed. If you stop to think whether or not your manipulations are likely to inflict pain or to excite or scare the animal, you should restrain it, but if the manipulation is not likely to cause pain or excitement, handle the cat in a quiet way and without haste. I know of no special method of handling cats analogous to that in dogs where we take advantage of hypnotics. I would, in conclusion, suggest careful attention to elimination in cats, this will forestall troublesome gastro-intestinal disturbances.

DR. MILLER: This morning somebody asked me about medication. I have made a habit of using an eye-dropper for any liquid I give in forced feeding. You can take a cat and go to a quiet place, place him on a table, play with him a little bit and turn his head back. Then take your eye-dropper, put it just back of the tusk, under the lip, and squeeze the bulb slowly. Ninety-nine times out of a hundred, I think, the cat will put his head up and will begin to lap. You can give medicine in the same way, if it is not bad to taste. You ought to give it

without any trouble whatever. It is the simplest thing in the world. The same procedure can be used in giving a pill. You can give a cat a pill much more easily than a dog. In doing that I generally take the head in my hand and bend it back. And another thing, do not try to have anyone hold the cat down and try to bend his head back into position. Set your cat up. All you have to do is straighten his head a little bit, press the cheek in, and drop the pill well back on the tongue.

A great mistake, I think, is made by many men in having somebody hold the cat down on the table. Set him up.

DR. J. A. CAMPBELL: A feature about feline practice is that it has its seasons. About the middle of January, it becomes very lively. The cat mating period seems to open officially with the new year. Young male and female cats that have just commenced to feel the urge of sex are presented for castration or ovariectomy, and the mature unsexed males come in suffering from all manner of injuries as a result of mix-ups while seeking the opposite sex. They receive scratches and bites on the head, fore-legs, different parts of the hind-quarters and tail. Many of these wounds are infected with *necrophorus bacilli*, which may require lengthy treatment.

In March and April, dystocia cases appear, and when the weather becomes warmer, skin diseases need attention and matted coats have to be unraveled among the Persians. Towards the end of the summer, infectious gastroenteritis plays havoc with the spring crop of kittens.

With regard to restraint: We use a canvas mat four feet square. It is of a light, strong, flexible material. This is spread out on the table, and the cat so placed on it that its head will protrude when the canvas is rolled up, and its body and claws securely confined, as in a strait-jacket, and no damage can be done with its claws.

Where there is anything that may require prolonged handling, such as surgery, treating wounds, removing mats from the coat, and the use of instruments in dystocia, we make it a practice to use a general anaesthetic.

In examining cats, we always inspect the ears carefully. You will find that almost all of the Persians have ear-mites. These can be demonstrated readily on a piece of white paper. Such a demonstration creates quite an impression on the owner, who has not suspected their existence.

With respect to unsexing cats: Occasionally you will have a male with only one testicle in the scrotum. The truant gland usually is located imbedded in an accumulation of fat in the inguinal region. There may be a little difficulty in the search if the subject is young and fat and the testicle undeveloped, in which case it is advisable to wait until the cat is more mature. If unsuccessful, you will have to invade the abdominal cavity to find the missing organ.

With regard to ovariectomy of the female, we have lost a great many in the past, so I determined to work out some method of reducing the mortality. We commenced with the median line opening and then went to the flank. It has many advantages over the former method.

By making a small opening into the abdominal cavity, using small hooks to pick up and extract the horns and ovaries, and not suturing the muscle wall, our mortality is now very nearly on a par with castration of the male.

We do not believe in keeping them in the hospital after the operation. They do far better at home.

Hematoma is a common occurrence in the cat and is a result of ear-mite infection. Before treating a hematoma, I always inform the owner that it may be a serious, stubborn condition to deal with, that it may take several weeks of treatment to effect a cure, and that the

ear may become deformed. I get that impressed on their minds at the start.

I find that the quickest results are obtained by keeping the incision as clean as possible and by using pressure by bandaging the ear over the top of the head.

I know of a practitioner that uses clamps on the ear, and he gets good results and very little deformity, but it is a difficult matter to keep any kind of a contrivance on a cat's head.

CHAIRMAN MORGAN: You mentioned the ear-mites being put on paper and shown to your client. Would you not prefer a microscope?

DR. CAMPBELL: You can readily see the mites with the naked eye.

DR. H. J. MILKS: I learned to spay both cats and bitches on the side. I got away from it for a while. I got back to it with cats. I did not have as good a reason as Dr. Campbell did. My reason was that we could send more cats home alive by spaying them through the flank, and that was all. I think that we almost always will have a little necrosis with the cat. We know of a good many men who are still spaying through the median line. They object to the side operation because they have to take off some hair. The reaction is that they would rather have the cat with the hair off than not to have the cat. The hair will grow back in a little bit, and our aim is to be able to send these cats home.

In regard to handling cats. I have always taken it as I did any other kind of work. Some of it is quite disagreeable. It used to be disagreeable in horses when you had to look after them pretty closely. We do not have any great number of cats in our neighborhood other than common cats. Most of them are pretty good. Once in a while, as in horse practice or anything else, you will find a tiger, and it does not take long to find that out. A veterinarian, to start off with, has to have a certain amount of self-preservation about him, and I notice that that develops with our students. After a cat has bitten or scratched some student, he learns that it may do it again. You can tell him about taking out the dog, that he has to grab him in the middle of the back. He will find out that we were not just telling him something to take up some time. A little experience in handling, a little perseverance, and the necessity for making a living will get you along.

As to food for cats, we have an old Tom cat which will go on a strike for a couple of days at a time if you do not give him what he wants to eat. He is boarding while we are away. I will wager that he eats but once in two or three days while we are gone, but he will be all right. If he does not get what he wants, he will go on a strike. They are very peculiar that way. We use ether and almost anything we want. We use bichlorid and alcohol for a disinfectant.

DR. J. E. CRAWFORD: I would like to find some antiseptic or disinfectant that will kill a Tom cat smell. Take a Tom cat, one or two or three years old, which is brought to the hospital. For some unknown reason some ladies will object to having him castrated. I can smell one when he comes in the office, and I have never been able to discover anything with which I can kill the odor of that Tom cat.

DR. R. J. GARBUTT: I was very much interested in hearing Dr. Campbell speak about hematoma. A great many people are under the impression that it is an abscess and that once it is treated you can take the cat home and the trouble is over. In view of the fact that it takes quite a while to cure, and a great many people do not care to spend too much money on a cat, particularly if he is an ordinary cat, I tried several different operations for hematoma. I anesthetize the cat and then I make a very small hole in the skin, possibly one-sixteenth

of an inch, and squeeze out all of the accumulated matter. Then I inject a small amount of tincture of iodine, seal the opening with a little collodion, and bandage it so that the cartilages are brought closely together. I change the bandage every day. In my first few cases I had very bad results, for I did not change the bandage for two or three days. As a result, the tip of the ear sloughed off. Sometimes I change the bandage twice a day and in most cases get very good results.

DR. H. J. MAGRANE: I had trouble in suturing in my operations. I used to have sloughing. Since I have used the sutureless operation, I do not have the trouble. I used to use one suture. Since using the sutureless operation, I have not lost a cat.

DR. G. A. H. EDMISTON: I would like to ask about the use of nembutal for cats.

DR. CRAWFORD: I have used it. A lot of veterinarians I know are using it all the time. It works very well.

MEMBER: In operating on cats and using nembutal, I find that it has worked very successfully. In fifteen minutes after giving it, any major operation can be done, and it produces a prolonged anesthesia in which one does not have to be too rapid in the procedure. I would much rather have it, for its facility and the impossibility of post-anesthetic mortality. I have used it in small cats as well as larger ones. I give it accordingly and never use less than a grain. I have had no mortality. I discontinued ether entirely with cats.

DR. J. L. RUBLE: I use 1 cc of procaine solution, injected in the scrotum. In three or four minutes you can scratch the animal on the back and he will purr. You can castrate him, and he will not move a muscle.

DR. D. A. PIATT: Have you tried nembutal intraperitoneally?

DR. CRAWFORD: Yes, sir, and it acts very satisfactorily.

### **Arsenical Sprays Endanger Consumers of Vegetables**

Nearly 1,500 crates of cauliflower containing residues of poisonous arsenical sprays, which growers in New York and New Jersey had shipped to dealers in Philadelphia, were condemned recently and destroyed by officials of the federal Food and Drug Administration.

It is necessary, for the control of destructive insect pests, to spray fruits and vegetables with lead arsenate and other harmful chemicals, but it has been proved that residues of such sprays may usually be removed by washing and trimming. The chances of foods carrying injurious residues can be minimized by following a judicious spraying schedule, according to federal officials. In recent years, most growers have learned how to remove residues of spray chemicals on fruits and vegetables, but there are still some producers who either through carelessness or ignorance neglect to make certain that their products are spray-free.

Over 200,000 dog tags, to be used in licensing dogs in 1933, are being distributed in Wisconsin.

## A LIVER FUNCTION TEST IN SHEEP\*

By J. N. SHAW, Corvallis, Oregon

Oregon Agricultural Experiment Station

The purpose of this paper is to deal with a dye test of the excretory function of the liver of sheep infested with *Fasciola hepatica*, the common liver fluke of sheep. Much has been written about the pathology produced by this parasite, but a good many questions still remain unanswered. Carbon tetrachlorid, the chemical used in treating sheep for this parasite, causes occasional losses. Lamson<sup>1</sup> and his co-workers have evidence to show that carbon tetrachlorid poisoning in dogs results from calcium deficiency, but this does not seem to answer entirely the question with sheep.

It was in the hopes of throwing some light on these phases of the fluke problem that led the author to try the liver function test. The test was used in a few other cases that will be included in this report. The use of dyes to determine excretory function of livers in humans and dogs is not new, but slight if any information is available as to the use of this test in other animals.

### METHOD

The dye used was rose bengal. Delprat, Epstein and Kerr<sup>2</sup> found that this dye is eliminated from the blood-stream of dogs and humans by the liver only. They also found it to be very non-toxic. The dye is very low in cost and in doses such as used in sheep does not produce any disturbance. Some hemolysis caused difficulty with the first tests, but this was overcome by using the dye dissolved in a 6 per cent dextrose solution.

The technic used was a variation in that used for humans and dogs by Delprat and Stowe.<sup>3</sup> The 1 per cent solution of the dye was made by dissolving the dye in a normal saline and adding enough dextrose to make a 6 per cent dextrose solution. When sterilized the solution is ready for use. Ten cc of this 1 per cent solution proved sufficient to color the plasma of the average-size sheep. This amount was quickly and easily injected into the jugular vein. Two minutes after injection, 10 cc of blood was taken, using a new syringe and a different needle each time. This sample of blood, mixed with 2 cc of a 2 per cent potassium oxalate solution, served as a normal. At this time the circulation was

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considered to have a maximum amount of dye in it. In 6 minutes from time the first sample was taken, or 8 minutes from injection, a second 10-cc sample was taken. These two samples, kept in a darkened container, were placed in the centrifuge as

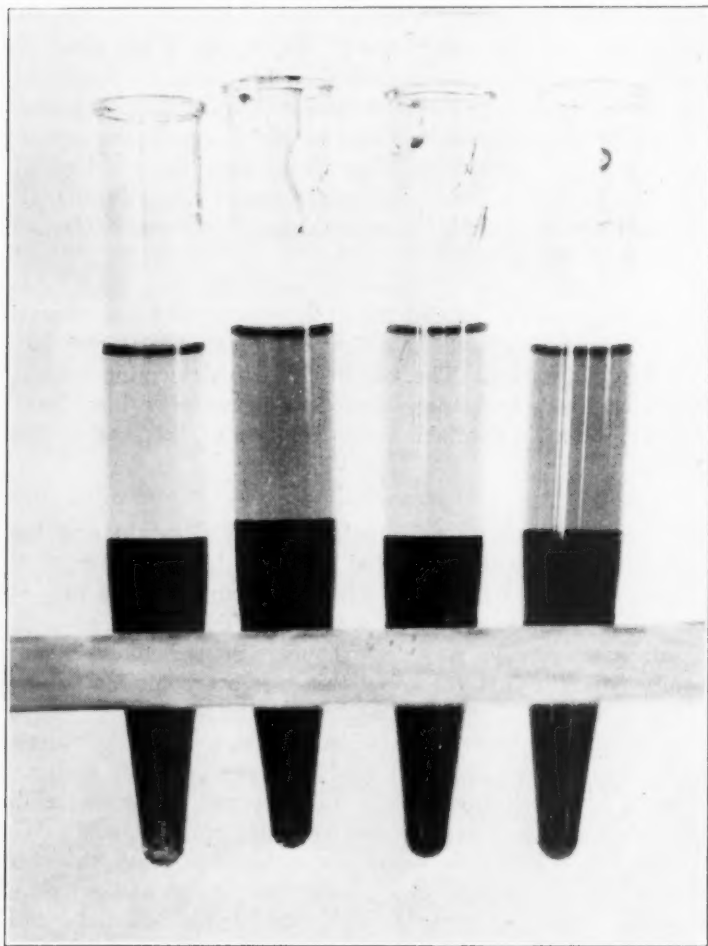


FIG. 1. Blood samples from two sheep. The darker tubes (2 and 4) contain plasma well colored with dye. Maximum amount of dye in plasma (2 minutes after injection). The lighter tubes (1 and 3) show how dye has been removed by the liver (8 minutes after injection).

soon after collection as possible. To one part of the supernatant plasma resulting was added two parts of normal saline. These

two solutions were then compared in the colorimeter. A 50 per cent elimination at 8 minutes indicated the normal function. Samples taken at 16 minutes and 25 minutes further indicated the excretory function of the liver, but in most cases were accompanied by hemolysis, which made comparisons difficult or impossible. An effort was made to overcome hemolysis by diluting the plasma with two volumes of clear acetone, as recommended by Delprat and Stowe.<sup>3</sup> This overcame the effects of hemolysis, but caused precipitation in plasma which, when centrifuged, carried the dye out of the plasma until comparisons were difficult or impossible. (See fig. 1.)



FIG. 2. Liver of sheep 5, from which 255 flukes were recovered. Fed 642 cysts on June 7, 1932, and killed on July 14, 1932.

For injection and collection purposes 18-gauge needles, one inch in length, with a 10-cc glass syringe, proved best. The plungers should be removed from glass syringes immediately after use to prevent sticking. In the rabbits used, 2 cc of a 1 per cent solution was injected into the vein in the ear and blood was collected direct from the heart.

In fat sheep with long wool, blood collections were not so easy, but no apparent harm resulted from many insertions of the needle into the jugular vein. Considerable blood collected under the skin in some cases but this disappeared in two days and, while the collection of blood from the jugular vein might have

been very painful, no evidence of pain was noticed after completion of the operation. In one or two cases the entire 10 cc of dye was injected under the skin but did not produce an abscess. The color of the plasma in some animals might cause some difficulty in making comparisons. In the case of the sheep, the plasma being practically colorless, comparisons were quite easily made



FIG. 3. Section of liver of sheep 5, showing one necrotic fluke burrow and one fluke, but no cirrhosis.

with the colorimeter and compared very closely with determinations made by using the spectroscope.

#### RESULTS

Fifty-six tests were made, 38 on supposedly normal animals and 18 on animals with some evidence of pathology of the liver.

Of the 18 abnormals, seven were passing fluke eggs in their feces, five had been fed viable cercariae and later three discharged ova in feces, two had been dosed with carbon tetrachlorid, three were suffering from so-called pregnant ewe paralysis and one, the rabbit, was discharging coccidia in the feces.

In the normals the colorimeter readings all indicated that at least 50 per cent of the dye was eliminated in 8 minutes and this was considered as indicative of 100 per cent excretory function of the liver.

In animals having some pathological condition of the liver, those having the light-colored livers quite characteristic of pregnant-ewe paralysis showed by the test a disturbance of the excretory function of the liver. These animals were in the later stages of the disease and would not have lived but a short time.

Sheep 5 showed a 30 per cent decrease in the excretory function of the liver. This sheep had been fed 642 *Fasciola hepatica* cysts five weeks previously. Gross pathology was produced, as shown by figures 2 and 3. This sheep was in excellent condition and did not show any symptoms that would indicate liver involvement.

Autopsies were had on 12 animals at varying lengths of time after dye injections. Six were normal animals or animals with apparently normal livers and six were animals suspected of having pathology of the liver. In the six normal animals, four sheep, one calf and one rabbit, the plasma showed 100 per cent liver function and autopsy not later than 45 minutes after the injection of dye revealed its presence either in the liver, the gall-bladder, or the first six feet of the duodenum. No dye could be found in any other organs of the bodies. In the six other animals (five sheep and one rabbit), pathology of the liver was suspected in three sheep because of symptoms of pregnant-ewe paralysis; in the two others, one because of dosing with carbon tetrachlorid and one having been fed fluke cysts; in the rabbit, because of large numbers of coccidia in the feces. The three pregnant ewes showed practically no elimination of dye as per colorimeter readings, and upon autopsy one showed dye in the duodenum and this only in very small amount. The case that had carbon tetrachlorid and the rabbit had 100 per cent liver function, dye being in the gall-bladder and the intestine of the sheep and in the liver of the rabbit. There was no dye in the intestine of the rabbit but the liver was markedly colored, which was accounted for by the killing of the rabbit 10 minutes after

injection. Sheep 5 showed only 70 per cent liver function, as a result of the pathology produced by the immature flukes.

#### DISCUSSION

No attempt is being made in this paper to advocate the use of this test in ordinary practice but since the test was first developed in dogs it would seem logical to see an opportunity to use this test as an aid to diagnosis in small-animal practice. It would seem also that this test has a place in our investigational work. Methods of checking the functions of other important organs are constantly in use, both in practice and research work.

Continued use of the test will bring about improvement in technic and more definite information as to the speed of this function in the livers of the different domestic animals. More accurate determinations of the amounts of dye present in plasma are apparently had by the use of the spectroscope, according to a personal communication from Dr. T. L. Althausen,<sup>4</sup> of the University of California Medical School. Spectroscopes and colorimeters are common instruments at most of our institutions where research by veterinarians is being done.

Authorities, in describing lesions of the liver in sheep produced by *Fasciola hepatica*, give cirrhosis a prominent place. Histological studies of such livers have failed to reveal cirrhosis and these findings are largely supported by the results of the excretion test. Althausen reports that "cirrhosis, whether of the portal or biliary type, always shows a reduction of dye excretion."

#### SUMMARY

1. The excretory function in livers of sheep infested with immature and mature *Fasciola hepatica* was not affected, according to the rose bengal dye test, except in one instance.

2. Carbon tetrachlorid in doses greater than necessary to kill immature and mature flukes did not produce sufficient damage to the liver to be detected by use of this test.

3. The excretory function of the liver of sheep suffering from so-called pregnant-ewe paralysis was markedly affected, as indicated by the rose bengal test.

4. The excretory function of the liver of the rabbit was not influenced by gross lesions of coccidiosis, according to this dye test.

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## SOME OF THE PRACTICAL PROBLEMS OF A POULTRY PRACTITIONER\*

*By H. J. SEAMAN, Wauseon, Ohio*

During the past decade, the veterinary profession has experienced many drastic and radical changes. Founded, and for a great many years developed and dependent, upon administering almost exclusively to the equine population, the science of veterinary medicine has undergone a complete metamorphosis. With the advent of a machine age, and with the constant migration from the rural communities to the various centers of population, there naturally has followed a corresponding increase in the demand for food, of which the domesticated food-producing animals have formed a considerable portion.

Likewise, with the growth and development of the automotive industries, mechanical creations have replaced the horse, until in some localities and in some lines of work the equine population has become almost extinct. The average practitioner of veterinary medicine has been forced constantly to readjust himself in order to keep abreast with his own practice and in some instances the changes have been so great and the readjustments so numerous, that it is not uncommon to hear our older colleagues speak of the first years of their professional careers as the "horse day."

During this period of readjustment, new phases of animal industry have been developed to take the place of the equine on the farm and at the same time furnish food products for the various non-food-producing occupations. In this connection, possibly no other phase of animal industry has made as much rapid progress as the poultry industry. Its growth and development has been almost phenomenal and, from an economic standpoint, it now ranks with any other source of farm income in the United States.

With the growth and development of this gigantic industry, we have witnessed the death-knell of the farm flock of a few years ago, tolerated because the owner was more or less of the opinion that a few hens were a necessary nuisance and composed of from 50 to 100 non-productive individuals of no particular breeding and for which very few provisions were made. The average farm flock of the present day is composed of from 200 to 500 well-bred, well-housed and scientifically-cared-for produc-

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tive hens, while in the larger commercial poultry-plants this number often runs into the thousands.

The element of unnatural environment enters into the practical side of the poultry industry in a great many instances. Especially is this true in regards to an unnatural and mechanical scheme of reproduction and to a program of stimulation, both in physical growth and in egg-production. From the time the egg is produced until the adult bird has outlived its usefulness, the life of the profitable hen is dominated by unnatural influences and unnatural environment. For instance, in the scheme of reproduction, the seasonal factors are totally disregarded in order to have pullets fully developed in time to produce winter eggs. Chicks are hatched in large numbers by means of artificial incubation, some are transported hundreds of miles when only a day old, artificially brooded, fed feeds rich in proteins in order to insure rapid growth and even are forced to submit to the regulation of their working day by the use of artificial light. This procedure is carried on generation after generation because of the impracticability of attempting to raise large numbers of chicks without the aid of artificial agencies. This endeavor to change the natural tendencies of the domesticated fowl, however, has brought the poultry industry face to face with some very confusing and perplexing problems, and perhaps the most confusing and perplexing of all have been the problems resulting from some disease. It is in this connection that the veterinary profession has been thrown into direct contact with the poultry industry of today and is becoming familiar with the phrase, "the poultry practitioner."

#### POULTRY INDUSTRY IN ITS INFANCY

First of all, we must realize the poultry industry is still in its infancy, and that amazing changes are constantly taking place. The veterinarian who is in any way interested in poultry practice and makes an honest effort to keep abreast with such a practice will realize this fact.

The amount of experimental work being carried on and the scientific reports being made, as a result of these experiments, perhaps can be understood more readily when we realize that at the present time there are 57 state and territorial agricultural experiment stations in the United States and in the territories governed by the United States, independent of the federal Department of Agriculture and its allied interests. To this mass of new information, we must add the scientific research being

carried on by the poultry departments of state agricultural colleges, state and private laboratories and by commercial laboratories interested in avian biologics.

Someone has said, "A text-book is always out of date," and never was this truer than when applied to the field of poultry. Pathology, preventive medicine, sanitation and hygiene, the use of biological products and diagnostic agents, and new methods of medication would be authentic for only a very short time in the field of avian medicine as we know it today.

This situation, coupled with the fact that in most instances the college training the average veterinarian received during his student days was very meager in regards to poultry diseases, only tends to bring us to the conclusion that to be well versed on the various branches of poultry practice calls for constant study and self-improvement. Therefore, it behooves every member of the veterinary profession who is at all interested in the field of poultry practice, to put forth extra effort in an honest endeavor to keep well versed with the new developments in this particular field and division of veterinary medicine.

State and federal bulletins are always available and no new bulletin should be published to which the veterinary profession should not have access, especially if it deals with something new in the field of poultry science. We, as individuals, should put forth extra effort to receive these new publications and, once received, they should be studied diligently.

#### POULTRY SUBJECTS GIVEN ATTENTION

Veterinary associations now list certain parts of their programs as devoted to poultry subjects and in many states the veterinary departments of the state universities and state agricultural colleges sponsor so-called short courses for veterinarians at which the latest developments in the field of poultry practice are considered. This necessity for self-education and study is characteristic of poultry practice, and constitutes one of the problems with which the profession has to contend. Anyone who is more than passively interested in poultry practice must be familiar with all phases of the poultry industry. He must understand, besides the disease problem, the principles of artificial incubation and brooding, the selection of breeding stock as to type and productivity, correct feeding and housing and the principles of sanitation and hygiene. Then too, the poultry practitioner must be diplomatic enough to sell his services.

Perhaps the most unfortunate factor encountered in a poultry

practice is the fact that the life of any single individual is of very little or no consequence. Both in farm flocks and in the commercial poultry plants, a certain death-rate is expected and tolerated, a condition we find existing in no other phase of animal industry. This fact in itself constitutes one of the necessities for and one of the problems of the poultry practitioner selling his services, and the old belief that a practitioner had nothing to sell but his services only tends to bring us to the realization that this is one instance where it is really necessary for a professional man to sell his services.

The obstacles encountered in this procedure constitute the majority of the practical problems of a poultry practitioner, since, after all, the practical problems of any business venture finally resolve themselves into financial problems influenced to some extent by volume and net profit.

The relative value attached to any individual tends to influence the poultryman in resorting to various practices before he seeks the aid of the veterinary profession in combating any disease condition. The poultryman must be met more than halfway in this case, and the quality of service rendered must be of such a high standard as to be desired even when other interests are willing to furnish free advice and free service. The type of service a practicing veterinarian is able and willing to furnish will be in direct correlation with the amount of service he is called upon to render, influenced to some extent by the amount of opposition encountered.

#### FORMS OF COMPETITION ARE MANY

The opposition to a poultry practice and the agencies furnishing such opposition are more varied and numerous than in any other field of veterinary medicine, especially from the standpoint of the practitioner. The poultry practitioner must sell his services in direct opposition to the diverse interests whose sole claim to recognition is the fact that they furnish and donate their services free of charge. It is possible in the majority of flocks to single out individual birds which are apparently unhealthy, thus making the poultryman a potential customer for every form of proprietary remedy. Also, the cost of treatment per bird is small, which in itself constitutes one of the strong selling points of every quack peddler who calls upon the poultry industry.

In times past, the equine practitioner had the quack to contend with, and regarded him as a menace to his practice, but for some reason or another, we readily tolerate the quack en-

countered in a poultry practice and have even given him the distinctive title of "poultry specialist."

One of the best examples of this practice is the commercial hatcheryman who refuses to cooperate with and recognize the veterinary profession. The average owner and operator of a commercial hatchery is first of all a business man who is conducting a private enterprise for the sole purpose of realizing a profit from his investment. Live baby chicks constitute his salable product and they are hatched and sold in such a way as to overcome competition, and at the same time induce the public to become interested enough to become the owner of this product.

Competition has become very keen between hatcherymen and, like any other business enterprise, they have been forced to rely upon advertising, salesmanship, guarantees as to the livability of their chicks for a certain period of time, and even promise free service on whatever chicks may be purchased. Naturally this background is built up for the prospective purchaser and after delivery has been made and some disease condition manifests itself, the local veterinarian is called in to witness the backfire of the forerunning sales propaganda. This often puts the veterinarian in a precarious position, because both the owner of the chicks and the hatcheryman may be good clients.

#### POULTRY CONDITIONS DEMAND SOUND JUDGMENT

There are many factors to consider before judgment is passed as to the cause of any given condition encountered in baby chicks. In most of his business transactions, the hatcheryman is forced to extend credit, so he naturally feels he has some right to make whatever suggestions he sees fit since he is still financially responsible for the chicks in question. A great many things are apt to happen to baby chicks during the first few days of their lives, over which the hatcheryman has no control whatever, yet the owner may feel the existing conditions are no fault of his, and endeavors to make the hatcheryman responsible if at all possible.

This again brings to mind the thought of unnatural environment and when we stop to consider the fact that mechanical appliances have been substituted for motherhood, that day-old baby chicks must live or perish on their own initiative, that they must learn to eat and drink without the aid of parental care or influence, that they must adapt themselves to artificial brooding and must even exist on foods largely unnatural, the thought

comes to mind that it is indeed a remarkable fact that the mortality in baby chicks is not greater than it already is.

A hasty diagnosis is detrimental to every party concerned, and never should be made. Disease may be present in day-old chicks or in week-old chicks, but this is not necessarily the fault of the hatcheryman. The poultry practitioner of a few years ago, who made only two different diagnoses, bacillary white diarrhea up until the chicks were three weeks old, and coccidiosis from then on, from necessity has passed out of the picture.

The veterinary profession has no bone of contention to pick with the modern hatching industry. There should be some sound business understanding between the hatching industry and the veterinary profession, governed by a code of ethics protective and beneficial to both interested parties. The ideal arrangement to furnish so-called free service on baby chicks is for the hatcheryman to advertise such free service and then employ his local veterinarian to render such service. The arrangement is apt to eliminate considerable friction between the hatching industry and the veterinary profession, and it tends to discourage the employe of the hatchery in question from being a so-called poultry expert to furnish such free service as may be needed.

#### A VALUABLE ASSET

One of the most valued assets in a private poultry practice is a sound business understanding with and the friendship of the local hatcheryman. This, however, at times may be impossible since certain hatcherymen are unwilling to coöperate in any sense of the word. Perhaps an example of this lack of coöperation will be more explicit.

In our county and in a neighboring town, we have a hatcheryman who has been established for fifteen or more years. He has always been at odds with the veterinary profession, due to his belief that there is no such disease as bacillary white diarrhea, and due to his belief that it is impossible to transmit any disease in a hatchery. This individual always has advertised free service on all baby chicks. He now has in his employ a young man whose education consists of a high school course, attendance at a farmers' week put on by the state university, and graduation from a short course of two weeks sponsored by the poultry department of the state agricultural college. This hatcheryman now advertises in the local papers to the effect that he has in his employ an Ohio State University trained expert, whose services are free at all times. He does not dispense any drugs

or practice veterinary medicine, but he does handle a complete line of poultry remedies which he prescribes.

This hatcheryman did confide in one of his colleagues to the extent that he was beginning to be of the opinion that infectious bronchitis might be transmissible through the hatchery, since he has not sold a single lot of chicks during the entire hatching season that was not affected to a certain degree.

We need coöperation with the hatcherymen who are willing to coöperate, but in some instances we need regulations to govern the hatching industry, not from the standpoint of financial gain to the veterinary profession, but for the protection of the poultry industry. In all other divisions of animal industry, certain regulations are enforced which tend to protect the industry itself, but in the case of the hatchery, we allow the very heart of the poultry industry to operate unlicensed, uninspected and in whatsoever manner it may see fit.

Surely the poultry industry is of sufficient value to deserve as much consideration as any other branch of animal industry. The agricultural interests also furnish a problem of considerable magnitude in regards to the opposition they furnish any practicing veterinarian who is more than passively interested in poultry practice.

#### AMERICAN PEOPLE "CHICKEN-MINDED"

The poultry industry owes a decided debt of gratitude to the extension departments of our state agricultural colleges and experiment stations, in that their activities have made the American people "chicken-minded." Free literature is available to whoever may request it, and due to the amount of this literature being distributed, more and more people are becoming interested in poultry farming.

The prevailing economic conditions have made it necessary for a great many people to seek other occupations, and there is being established a "back to the farm" movement over the entire nation. This movement has terminated in the establishment of numerous poultry farms, since in this type of farming a permanent start can be made in a few months and the salable products always find a ready market. With some study and with the free aid furnished by the agricultural interests almost anyone can be reasonably sure of becoming capable of caring for a farm flock in such a way as to realize a net profit.

In this same connection, the veterinary profession also owes a debt of gratitude to the agricultural interests, in as much as

poultry practice has been developed only to keep pace with the growth of the poultry industry. However, the practicing veterinarian has at times felt the effects of some of the policies and projects sponsored by these same agricultural interests.

Free bulletins dealing with poultry diseases have become so numerous and so specific in their discussions of poultry diseases as to become a form of governmental competition. The authors of these bulletins often are members of the veterinary profession and they are to be complimented on the type of information contained in these free bulletins. The scientific research carried on by these members of our profession has been of untold value to the poultry industry as well as to our profession. They should be complimented on the clear and concise manner in which they have handled their subjects.

But some of these free bulletins dealing with poultry disease are in every way a short course in veterinary medicine, intended for the average poultryman, and contain more scientific and technical information regarding the treatment of poultry diseases than was available in the entire curriculum of the veterinary college of a few years ago.

#### VOLUMINOUS LITERATURE ON POULTRY SUBJECTS

There is no regularity concerning the policies followed by the several state and federal departments of agriculture in regards to the amount or the type of information published. There is also no similarity concerning the nature of this information. One agricultural college, located in one of the eastern states, lists for distribution 115 bulletins dealing with poultry subjects and 31 of these bulletins deal specifically with some poultry disease.

Do you realize that there are in the neighborhood of 600 bulletins now published by our experiment stations under the classification of poultry and that over one-third of these bulletins deal with poultry diseases and, with the exception of several states, treatments are readily discussed and described? Is it any wonder we must compete with poultry specialists on every hand, the commercial hatcheryman, the commercial feed salesman, the county agricultural agent, the quack peddler with some sure cure, and even the graduates of farmers' weeks and culling schools sponsored by our state agricultural colleges? A little knowledge is a dangerous thing and unless we as a profession begin to realize this fact, the veterinary profession faces a crisis.

Is it logical to be forced to compete with everyone who cares to take advantage of the fact that a rather liberal education

on poultry diseases is available free of cost? Shall we, as a profession, turn our heads and refuse to believe, while we drift back to the standards of the "horse doctor" days?

From a specific standpoint, I wish to cite several examples which will tend to demonstrate the degree of consideration and coöperation extended to the veterinary profession by the state departments of agriculture, as well as the federal Department of Agriculture:

*Bulletin 1:* A 62-page bulletin, dealing with diseases and parasites of poultry and containing a description of 43 diseases and parasitic conditions commonly encountered in a poultry practice. Each condition is described as to symptoms, cause, postmortem appearance (which is equivalent to a description of the diagnosis and differential diagnosis of disease), prevention and treatment.

In the introductory remarks there appear the following paragraphs:

The aim in studying diseases of poultry is therefore to learn how to prevent as well as how to cure them. The treatments recommended in this bulletin are in accordance with the best present knowledge of the subject. However, methods of controlling certain diseases are not yet based on experimental procedure, but investigations to obtain such scientific information are in progress.

Here we have a free bulletin, dealing with all the common pathological conditions encountered in poultry, in which the veterinary profession is completely ignored. Not once, in the entire discourse contained in this bulletin, is the poultryman advised to consult his veterinarian.

*Bulletin 2:* A bulletin entitled, "Common Diseases of Poultry," which utilizes the first and most prominent page in explaining in bold type the necessity of and the correct procedure to follow in sending any and all sick birds to the poultry department at the state agricultural college for diagnosis.

Here are a few of their recommendations:

Curative medicine in most cases has little place in poultry work, due to the short span of life of the individual, to the low monetary value of each bird and to the time consumed in treatment. Such a treatment has a poor chance of success.

Under the heading, "General Prevention of Disease," the bulletin states:

The systematic use of purgatives is helpful in preventing disease and should be given at least once every three weeks at the rate of one pound of Epsom salt to each one hundred birds. This may be given by dissolving the salts in the drinking water. Sodium hypochlorite solutions such as commercial B. K. Compound at the rate of one tablespoonful to the gallon, or bichloride of mercury at the rate of one tablet to two gallons of water, are excellent germi-

cides and should be used whenever any contagious diseases appear in the flock.

Twelve common diseases are described and this treatment followed rigidly with only a slight deviation in the amount of Epsom salt recommended. The treatment for respiratory diseases and fowl-pox (which is described as "a disease caused by a germ and manifested by wart-like nodules") consists of the use of a spray composed of twelve tablespoonfuls of B. K. to each gallon of water. The discussion of vaccination in order to prevent pox ends by stating that "material for vaccinating may be secured from any reliable biological house or from the local supply dealer. The expiration date of the vaccinating material may be noted on the bottle."

Roup is described as "a disease supposed to be caused by a very small germ and the recommended treatment is one pound of Epsom salt to each 400 pounds of chicken, and the use of B. K. in the drinking water and as a spray." Evidently the poultry industry might suffer severely in this state if the manufacture of B. K. ever were discontinued.

*Bulletin 3:* A 72-page bulletin constituting a review of the experimental work and the recommendations of ninety-two different authors. The bibliography contains mention of articles appearing in the JOURNAL of the American Veterinary Medical Association, no fewer than eight times; "Poultry Diseases," by B. F. Kaupp; "Diseases of Domesticated Birds," by Ward and Gallagher, and the mention of almost every member of the veterinary profession regarded as outstanding in the field of poultry science.

#### DIAGNOSIS SIMPLIFIED

The diagnosis and differential diagnosis of poultry diseases are handled in a very novel manner. The symptoms simply are listed and grouped according to visible pathological lesions occurring in any portion of the body, followed by the logical diagnosis.

As an example, the pathological changes of the liver are divided into "enlarged, white spots and yellow." An enlarged liver points to fowl typhoid, leukemia, blackhead, or apoplectic form septicemia. White spots limit the diagnosis to blackhead, coccidiosis, tuberculosis and fowl typhoid, while the yellow liver may be caused by chilling, overheating or by sand scours. This procedure is followed in the description of 25 different portions of the body and 56 specific disease conditions. Several pages are devoted to correct postmortem technic and the bulletin con-

tains 65 illustrations. The appendix consists of a table of approximate equivalents, listed in such a way as to be reckoned in teaspoonful and tablespoonful doses, a table of percentage solutions and a so-called dosage table, in which 56 of the more common drugs are listed, with the therapeutic, non-toxic and toxic dosages.

The writer, who, by the way, is connected with the poultry department of a state agricultural college, sums up the purpose of this bulletin by saying:

The purpose of this bulletin is to aid poultrymen in preventing and controlling diseases in their flocks. The information is based on a summary of the writings, lectures and personal opinions of the best-known authorities on poultry diseases. The bulletin has been prepared in response to urgent requests of many poultrymen for some source of practical information on the prevention and control of poultry diseases.

He should have added further and admitted that it was published at the request of would-be poultry specialists of his state as an abbreviated handbook for the use of poultry quacks, condensed from scientific publications and simplified to the extent that they might at least partially understand it.

*Bulletin 4:* A very technical bulletin on the subject of bacillary white diarrhea or pullorum infection of the domesticated fowl. This bulletin contains a result of the experimental work conducted over a period of years and contains twenty tables of results obtained in these experiments. The summary of results is very clear and concise, and the information contained therein is of untold value to both the poultry industry and the veterinary profession. The suggestions for the control of the disease include blood-testing. Bulletins of this nature are of far more value to all concerned than the aforementioned bulletins.

*Bulletin 5:* Here is a bulletin published on "Keeping the Farm Flock Healthy," and it contains a great deal of practical information for the poultryman. It is also a step in the right direction, from the standpoint of the veterinary profession. Here is an example of the sensible recommendations appearing in this bulletin, under the heading, "How to Handle Disease":

In case disease appears in the flock, the first essential is a correct diagnosis. In recent years numerous veterinarians of this state have received special training in the diagnosis and control of poultry diseases. These men are in a position to give expert diagnostic service. Take typically affected specimens to them for autopsy. After the nature of the disease has been established, consult them with regards to methods of treatment. Qualified veterinarians should always be employed for the application of the tuberculin and pullorum-disease tests.

Bulletins of this nature will cost the poultry industry fewer losses and effect a decided saving in money, and for the good of the industry itself more information of this sort should be distributed, instead of the average bulletin published by the majority of agricultural experiment stations.

Would it not be a better policy to put every licensed veterinarian on the mailing lists of the agricultural experiment stations and furnish them with scientific information they are able to understand and utilize, than to oppose the profession by attempting to furnish free information on the treatment of disease conditions to whoever is able to read it? Whenever any broadcast is sponsored by the Department of Agriculture, such as the "National Farm and Home Hour," the broadcast always ends with the recommendation of a certain farmers' bulletin, published by the United States Department of Agriculture and free upon request. Can the A. V. M. A., through combined effort and influence, change the ending of these broadcasts to the extent that, when recommendations are in order, the veterinary profession will be recommended?

Such recommendations are generally made concerning the dental and medical professions. Their members, through ethical, legitimate advertising, coöperation and organization, have developed their professions into profitable business ventures. These same business ventures by the same coöperation and organization, are guarded zealously from any unethical competition.

On August 1, there appeared in the daily press a news item under the heading, "Medical Group Plans Protest":

Spokesmen for the American Medical Association will appear before the Shannon House Committee when it resumes hearings here Thursday, August 4, to complain against government competition. The medical association contends disabled war veterans can be treated more effectively and at a great saving of money by physicians outside government employ. The organization's complaint will be voiced by Dr. E. S. Skinner. Veterans of Foreign Wars, the American Legion, and the Disabled American Veterans, presumably advocates of the present system of hospitalization, will be given the opportunity of replying to Dr. Skinner next Friday.

This is a typical illustration of the constant warfare waged by the American Medical Association against any form of unethical competition. Then, too, the medical and dental professions have realized the possibilities of ethical advertising.

Surely all of us are familiar with the most popular form of advertising ever sent out by the National Broadcasting Company, and we are equally familiar with the slogan accompanying this advertising, "and see your dentist at least twice a year."

This form of advertising has not only made a certain brand of tooth paste universally popular, but it has been worth thousands and thousands of dollars to the dental profession from a financial standpoint.

The medical profession is even more fortunate in the type of distinguished advertising it receives. In almost every issue of the leading periodicals there appear full-page advertisements sponsored by some interest remotely connected with the medical profession, but intended nevertheless to advertise the medical profession. In the August issue of one of these periodicals there appeared several advertisements of this type.

One was the advertisement of a nationally known biological concern and was entitled, "Elbert Hubbard's Idea of Wealth." It read:

If you have health you probably will be happy, and if you have health and happiness, you will have all the wealth you need even if not all you desire.

Following this quotation but none the less prominent, the advertisement continued:

If you have good health, protect it at all times. It is far easier kept than reclaimed once it has passed. Have a health plan. Go to your physician at regular intervals for a careful physical examination, and call him promptly whenever illness threatens.

In this same periodical, there appeared two other full-page ads complimenting the medical profession and advocating long life and happiness by keeping physically fit by means of regular physical examinations. One of these was sponsored by a nationally known drug company and the other by one of the largest life insurance companies in the world.

Surely this type of advertising tends to produce a beneficial effect upon the medical profession, both from a practical and financial standpoint. Is it not possible for the veterinary profession to follow such a procedure in selling our profession to the public, the same as the medical and dental professions have been sold? Will not sincere coöperation and organization bring about a like state of affairs and tend to make the veterinary profession more respected and demanded? Surely the results will justify the effort, and the average practitioner of veterinary medicine will find his practical problems, to a large extent, have disappeared.

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"Which is correct? The hen is sitting or setting?"

"Why worry about that? It's more important to know whether she's laying or lying!"

## A SPECIFIC INFECTIOUS DISEASE OF CHICKENS DUE TO A HEMOLYTIC STREPTOCOCCUS\*

By C. B. HUDSON,† *New Brunswick, N. J.*

*New Jersey Agricultural Experiment Station*

On February 24, 1929, Mr. Lamb presented at the laboratory the bodies of two White Plymouth Rock hens for postmortem examination. The birds came from the farm of Mr. R., of Swedesboro, N. J. The history obtained at that time was rather incomplete. Mr. Lamb stated that Mr. R. had lost a number of birds recently. The birds died suddenly and without apparent cause.

Postmortem examination of the two birds revealed practically the same lesions. There was a hemorrhagic discoloration of the breast muscles. The body cavity contained a small amount of reddish fluid. The liver and spleen were enlarged and showed small pin-point necrotic foci. The lungs were slightly congested, the intestines were catarrhal and hemorrhagic, and finally, a small amount of reddish fluid was found in the pericardial sac. Blood cultures were made on agar plates, and after incubation for 24 hours at 37° C., the plates might easily have been discarded, since the growth was so slight as to be almost invisible. However, with the aid of a hand lens, numerous, very small pin-point colonies could be seen. After incubation for 48 hours, the colonies could be seen easily with the naked eye.

Broth cultures were prepared from the plates and a preliminary inoculation made into two birds by the intravenous and subcutaneous routes. These sickened in 48 hours, and died in 7 and 8 days, respectively. Blood cultures made from these birds were positive. Stained blood-smears revealed the causative organism to be a short-chained streptococcus.

### HISTORICAL

A review of the literature reveals the fact that only a few outbreaks of streptococcic septicemia have been reported in fowl.

In 1902, Norgaard and Mohler<sup>1</sup> reported on a highly fatal septicemia of fowl due to a non-pyogenic streptococcus. The outbreak occurred on a farm in Loudoun County, Virginia. During the course of the disease, which lasted practically four months, about 220 birds out of 240 succumbed. The source of the infection was never discovered. No birds had been purchased except from two adjoining farms, where the stock was said to

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†With the coöperation of G. E. Lamb, County Agricultural Agent, Gloucester County.

be healthy. Norgaard and Mohler determined the causative organism to be a short-chained streptococcus. A bacteriological and pathological study was made, and the pathogenicity of the organism determined for several species. Rabbits, pigeons, and ducks as well as chickens succumbed after inoculation. Ducks, however, appeared to be less susceptible than chickens. The disease was given the name, "apoplectiform septicemia."

Dammann and Manegold<sup>2</sup> reported, in 1905, on a disease of fowl produced by a capsulated streptococcus. They named the causative agent *Streptococcus capsulatus gallinarum*. The disease was said to run a chronic course. The outbreak lasted about 3 months, and only 8 birds out of 100 were lost. Experimentally inoculated birds showed considerable variation in susceptibility, some birds lived only 7 days after inoculation, while others succumbed only after 30 to 50 days. They found that the disease could be transmitted by subcutaneous inoculation, or by the inhalation of the bouillon cultures. Pigeons, white mice, rabbits and lambs were found to be susceptible, while dogs, ducks and guinea pigs were refractory. The source of the infection never was discovered. The hens on an adjoining farm never were affected. These were said to be at a distance of about 50 feet. The most characteristic symptom observed was a more or less pronounced sleepiness of the affected birds. The disease was termed "sleeping sickness."

In 1908, Greve<sup>3</sup> described a similar infection of fowls. The causative agent, a capsulated streptococcus, was assumed to be the same as the *Strep. capsulatus gallinarum* of Dammann and Manegold. He found, however, that the course of the disease was very acute, that the birds died suddenly in one-half to one day after the onset of symptoms. This is in contrast to the more chronic type of the disease as observed by Dammann and Manegold. The source of the infection never was determined. However, Greve noted that only Minorca pullets, which had been reared from hatching eggs the previous spring, were affected while the adult stock with which they associated was not attacked.

In 1910, Magnusson<sup>4</sup> reported on a highly acute outbreak of streptococcus infection in chickens. In 14 days, 129 out of 202 birds died. The disease spread to neighboring farms and 178 birds were lost out of 587. In addition to Plymouth Rocks and White Leghorns, there were two native races on the farm. The Leghorns appeared to be more susceptible than the Rocks. On the other hand, the native races were very resistant, and only a few of them died. A Gram-positive streptococcus, occurring

in the blood and organs in short chains, was determined to be the causative organism. Bacteriological and pathogenic studies of the organism were made. Mice, pigeons and rabbits in addition to chickens were found to be highly susceptible. Sparrows, cats, dogs, horses and cows were found to show considerable natural resistance. Guinea pigs and young pigs were found to be refractory. A dog which was inoculated intravenously became lame in the left foreleg one week later. An abscess formed at the knee from which the organism was isolated. This was the only experiment that resulted in suppuration and localization. In all other experiments the streptococcus was non-pyogenic.

By serial inoculation from rabbit to rabbit, and from bird to bird, Magnusson obtained two forms of the organism. The rabbit strain would no longer affect birds. By a similar experiment with mice, he obtained strains which would no longer affect rabbits. No capsule formation was observed as in the outbreaks reported by Dammann and Manegold and by Greve. Because of the absence of apoplectiform symptoms and the subdural exudate described by Norgaard and Mohler, Magnusson considered the expression "infectious streptomycosis of hens," a term suggested by the veterinarian Sven Wall, more appropriate than apoplectiform septicemia as a name for the disease. The source of the infection never was discovered, although Magnusson stated that the disease probably was due to infection with an otherwise avirulent strain which had become virulent.

#### HISTORY OF THE OUTBREAK

After having isolated a streptococcus from the first cases, Mr. Lamb was requested to send in more specimens, and to obtain a more complete history of the outbreak. Unfortunately, as far as this study is concerned, only one more bird died (March 19), and from it a streptococcus was isolated. Although Mr. Lamb had been requested to obtain a complete history of the outbreak, it was deemed advisable to make a personal visit to the farm on which the outbreak occurred. Unfortunately, this visit was not made for some time after the losses had stopped, but the following history was obtained:

At the time the disease appeared, the flock consisted of 250 White Plymouth Rock hens and pullets. Of this number, all but about 25 were kept in a small house located about 150 feet from a barn which housed the smaller flock. The larger flock was allowed free range in an enclosure which separated it from the smaller flock. The smaller flock ranged around the barn

and roosted there. According to Mr. R., the disease made its appearance in the larger group of birds about the middle of November, 1928, and lasted until about the middle of March, 1929. During this period, 125 birds were lost out of the 225 comprising the larger group. No losses occurred in the 25 birds, nor did any of them show any indication of being affected. The only introductions made into the flock were two cock birds. These were purchased from a neighbor on November 1, and placed in the larger flock. At this same time an epizootic of colds was in progress in the larger group of birds. One of the cock birds, which had been ailing for some days, died two weeks after its purchase. The bird was not examined, so that the cause of its death is unknown. Within a few days, however, deaths occurred among the pullets. No old birds were lost until the latter part of December. The neighbor from whom the cock birds were purchased experienced no unusual losses in his flock.

According to Mr. R., some of the birds were found dead on the roost or around the yard without having shown any sign of illness. Others would linger for days, standing in a hunched-up position with their eyes closed as if asleep. As far as Mr. R. was aware, no birds recovered.

In the early spring of 1929, hatching eggs were collected from the smaller flock housed in the barn. They were hatched and brooded by hens from the same group. The mortality in the chicks was unusually low. At this time Mr. R. had not deemed it advisable to hatch eggs collected from the diseased flock. However, after the losses stopped it was considered safe to hatch eggs collected from the survivors. Accordingly, eggs were collected, hatched and brooded by hens which had apparently escaped the infection. The chicks were brooded in the coop in which the outbreak occurred. Before placing the hens and young chicks in the house, the latter was thoroughly disinfected and about three inches of the soil removed and replaced with fresh soil. Two hundred chicks were placed in the house to be brooded by ten hens. Mr. R. stated that practically all the chicks in this lot died of what appeared to be the same disease which had affected the adult stock. The losses started after the chicks were two weeks old, and continued until the chicks were about five weeks old. Some of the chicks died without showing any previous symptoms, while others lingered for some time and presented the same symptom of sleepiness that was observed in the case of the adult birds.

### SYMPTOMS

The symptoms described by the owner closely paralleled those seen in inoculated birds. Inoculated birds may die within 24 hours after inoculation without showing symptoms. In less acute cases, the bird presents an appearance of extreme depression. The feathers are ruffled, the eyes remain closed as if the bird were in a deep sleep, and are opened only momentarily when the cage is approached. Very little food and water are taken. In the majority of cases death takes place in from one to two weeks. However, two birds were observed to live 73 and 78 days, respectively.

### POSTMORTEM

The usual postmortem picture is as follows: Mucous membrane of the head pale. Hemorrhagic discoloration of breast. Liver enlarged, showing pin-point necrotic foci and covered with a clear plastic exudate streaked with extravasated blood. The spleen and kidneys are swollen. The intestines are catarrhal and hemorrhagic. The lungs show circumscribed areas of congestion. The body cavity contains a small amount of reddish fluid. The heart muscle shows hemorrhagic discoloration, and the ventricles contain an abnormal amount of discolored serum. The pericardial fluid is reddish in color. The postmortem findings paralleled very closely those observed by Norgaard and Mohler except for the absence of any large amount of exudate in the abdominal cavity.

### BACTERIOLOGY

The organism usually may be isolated in pure culture from the heart or any of the organs. Stained smear preparations made from the blood, bone-marrow and various organs, reveal the presence of a short-chained streptococcus varying in the number of elements from 2 to 20. Chains of 6 to 8 elements predominate.

### MORPHOLOGY

The morphology of the organism resembles very closely that described by Norgaard and Mohler. There is a great diversity in the number of elements appearing in a chain, depending on the medium used for cultivation. In the blood and tissues the chains are short, and contain from 2 to 20 elements. Chains of 6 to 8 elements predominate. The chains usually contain an even number of cells. In certain fluid media the organism appears in chains of over 100 elements. This characteristic is best demonstrated in 1 per cent dextrose broth. On solid media the organism occurs as a diplococcus. Cells twice the ordinary

size frequently are seen. These occur either in the middle, or at the end of the chain. Involution forms are found in old cultures.

The organism is Gram-positive and also stains with Wright's stain and the ordinary aniline dyes. It is non-motile and no capsule has been observed.

#### CULTURAL CHARACTERISTICS

*Agar:* On ordinary beef extract agar adjusted to a pH of 7.2 the organism grows very slightly or not at all. However, when cultures are taken from the blood and put on this medium, very fine pin-point colonies may be seen after incubation for 48 hours at 37° C. The blood undoubtedly stimulates growth of the organism.

*Starch agar:* On this medium, or agar to which 1 per cent dextrose has been added, small, white, convex colonies develop in 24 hours at 37° C. They do not tend to coalesce. After 48 hours they appear to have a brown center surrounded by a pale bluish border.

*Blood agar:* Agar containing 5 per cent whole chicken blood gave excellent growth in 24 hours at 37° C. On this medium the colonies are well isolated, white in color, and convex. They are smooth, shiny by direct light, and frequently gelatinous in consistency. After incubation for 48 hours, they appear dull by direct light and have a brown center surrounded by a pale bluish border. By this time they have become granular. The colonies reach their maximum size in 72 hours. A zone of hemolysis about 3 mm. wide surrounds the colony. Cultures carried on this medium and transferred, every two or three months, retain their virulence for at least a year. Blood agar, to which 1 per cent dextrose has been added, gives even better growth, the colonies reaching a diameter of 5 to 6 mm. with a very wide zone of hemolysis.

It should be noted that Norgaard and Mohler make no mention of hemolysis by the organism studied by them. It is to be assumed, however, from the postmortem lesions which they noted, that the organism did possess this property. Magnusson, on the other hand, noted hemolysis in blood plates prepared from ox blood. He did not, however, study the organism on blood plates made from whole chicken blood.

*Broth:* The organism grows only sparsely in ordinary beef extract broth adjusted to a pH of 7.2. However, if 1 per cent dextrose is added, a very profuse growth takes place in 24 hours at 37° C. The organism develops in long chains at the bottom

and along the sides of the tube, thus forming skeins or flocculent masses, while the medium itself remains clear. In a few days, the growth settles to the bottom, but on shaking, the tube breaks up into numerous small particles without causing a uniform turbidity of the fluid.

*Blood broth:* The organism grows well in blood broth prepared by adding 5 per cent whole citrated chicken blood to ordinary beef broth. The added blood, however, apparently prevents the formation of long chains. When grown in this medium, short chains are formed such as those seen in the blood and organs of birds affected with the disease in question. In whole chicken blood the organism also grows well and only short chains are observed.

#### FERMENTATION REACTIONS

The reactions of the organism in 22 substances were determined. One per cent solutions of these were prepared in Dunham's peptone water containing Andrade's indicator. Five-hundredths of 1 cc of whole citrated chicken blood was added to each tube just before inoculation to insure growth. After 48 hours, a blood-agar culture was made from each tube to show that growth had taken place. The fermentation tubes were incubated for two weeks. At this time each tube was cultured again to determine whether or not contamination had taken place. Tubes which showed acid at the beginning of the experiment were found to be sterile. Dextrose, levulose, lactose, maltose and saccharose were attacked in 24 hours; dextrin and soluble starch in 48 hours; galactose and salicin in 4 and 6 days, respectively. In no case was gas formed. Arabinose, xylose, trehalose, raffinose, melizitose, inulin, rhamnose, adonitol, dulcitol, erythritol, inositol, mannitol and sorbitol were refractory.

Norgaard and Mohler found that their organism fermented dextrose, lactose and saccharose with the formation of acid but no gas. None of the other sugars were used. Magnusson tested his organism on glucose, galactose, saccharose, lactose, maltose, mannitol, dulcitol and sorbitol. A one per cent solution of each of these substances was prepared in Cibil's meat extract bouillon. Magnusson reported acid in all of these but no gas formation. It is quite likely, however, that at least some of the fermentations which he observed were non-specific, since meat extract medium easily may contain some sugar.

#### ANAEROBIC GROWTH

Cultures of the organism were placed in a tightly closed jar and sealed with paraffin. The jar then was attached to an

electric vacuum-pump and the air exhausted. The organism grew as well here as under strictly aerobic conditions.

#### BIOLOGY

The organism is an aerobe and a facultative anaerobe. It produces hemolysin and grows very poorly on ordinary beef-extract agar or in beef-extract broth. It grows well, however, on media to which whole chicken blood has been added, or to which has been added any of the sugars which are readily attacked. When kept on whole chicken blood-agar media, and transferred every three months, the organism retains its vitality as well as its virulence for at least a year. Its optimum temperature is 37° C., although it will develop slowly at room temperature.

#### PORTAL OF ENTRY

Magnusson made no attempt to determine the portal of entry of the streptococcus which he studied. Norgaard and Mohler, on the other hand, reported on feeding experiments with their organism and leave the impression that they considered the portal of entry to be the intestinal tract. In their experiments six chickens which had fasted for 24 hours were fed a few cubes of bread soaked in a fresh bouillon culture. This was repeated for three consecutive days. Death of the chickens followed in 4 out of 6 cases in from 4 to 13 days. At a later date, two remaining birds were given a small dose of culture intravenously, and both succumbed to the disease. The incubation period, however, was longer than that experienced in other birds similarly inoculated. No doubt these birds either possessed some natural immunity or had acquired some immunity as a result of the heavy feeding of the organism. No mention is made of the fact that, although the dose used in the experiment was many times greater than a bird would ever receive under natural conditions, the incubation period was greatly extended beyond that seen in the natural disease.

Dammann and Manegold found that the disease studied by them could be transmitted to other hens by subcutaneous inoculation of fragments of the organs or culture suspensions. Inhalation of bouillon cultures also gave positive results. They were unable to reproduce the disease by the feeding of liver or blood. It is remarkable to note that they were not able to reproduce the disease by intravenous inoculation.

The purpose of the following experiments was to determine the natural portal of entry of the organism. Accordingly, 13 birds were inoculated intranasally, and 14 birds per os. Intra-

nasal inoculations were made by dropping 0.2 cc of a 24-hour blood-broth culture into the nasal cleft. All inoculations per os, with the exception of birds 3 and 4, were made by giving a gelatin capsule containing culture with the aid of a capsule-gun. The capsules were released in the crop, care being taken not to contaminate the nasal cleft or larynx. Three-tenths of 1 cc of a 24-hour blood-broth culture was used in all inoculations per os. The results are shown in table I.

TABLE I—Results of various methods of inoculation.

BIRD	METHOD	DOSE (cc)	DATE OF INOCULATION	RESULT
1	Intravenous	2	3-8-29	D. 3-15-29
2	Subcutaneous	2	3-8-29	D. 3-16-29
3	Drinking water	5	3-26, 3-29 and 4-2-29	Lived
4	Feed	5	3-26, 3-29 and 4-2-29	Lived
5	Per os	0.3	3-26-29	Lived
6		0.3		Lived
7		0.3		Lived
8		0.3		Lived
9		0.3	4-12-29	Lived
10		0.3		Lived
11		0.3		Lived
12		0.3		Lived
13		0.3	4-19-29	Lived
14		0.3		Lived
15		0.3		Lived
16		0.3		Lived
17	Intranasal	0.2	3-26-29	D. 3-29-29
18		0.2		D. 4-10-29
19		0.2	4-12-29	D. 4-16-29
20		0.2		D. 4-13-29
21		0.2		D. 4-23-29
22		0.2		D. 4-21-29
23		0.2	4-19-29	D. 6-24-29
24		0.2		Lived
25		0.2		D. 4-20-29
26		0.2		Lived
27		0.2		D. 7- 6-29
28		0.2		D. 4-28-29
29		0.2		D. 5- 7-29

Table I shows very clearly that the natural portal of entry of the organism is by way of the respiratory tract, and that the disease cannot be produced when inoculation per os is carried out in such a way as to prevent contamination of the respiratory tract. The table shows also the variation in susceptibility among the birds inoculated. Some birds succumbed in 24 to 72 hours following intranasal inoculation, while in two cases death did not take place for 72 and 78 days, respectively. Two birds were

not affected by intranasal inoculation and were apparently naturally immune. These birds will be discussed later. The results also show that the disease is always fatal. That the disease cannot be readily reproduced by giving a comparatively large dose of the organism in the food and water is shown in the cases of birds 3 and 4. These birds were shown, by subsequent intranasal inoculation, to be susceptible to the disease. It is to be admitted, however, that in consuming feed or drinking water in which the organism is present, the nasal cavity may become contaminated and disease may result. This probably accounts for the successful feeding experiments reported by Norgaard and Mohler.

In order to show that birds which resist infection per os are susceptible to the disease, birds 4, 5, 6 and 7 (table I) were given 0.2 cc of a 24-hour blood-broth culture intranasally. The results are shown in table II.

TABLE II—*Results of intranasal inoculation of birds which resisted inoculation per os.*

BIRD	DOSE (CC)	DATE OF INOCULATION	RESULT
4	0.2	4-12-29	D. 4-13-29
5	0.2		D. 4-14-29
6	0.2		D. 4-24-29
7	0.2		Lived

Table II shows that birds 4 and 5 were highly susceptible to the disease, death taking place in 24 and 48 hours, respectively. These birds previously had resisted a dose of 5 cc of a 24-hour blood-broth culture given in the feed and water on three occasions at intervals of three days. Of the two birds (6 and 7) which had previously been inoculated per os by means of a capsule, only bird 6 succumbed to intranasal inoculation. Bird 7 was evidently naturally immune and will be discussed later.

#### THE CARRIER STATE

The following experiment was carried out in order to determine the possibility of the existence of a carrier state among birds that resist intranasal infection. Accordingly nasal cultures were made, at intervals, of all the birds which showed marked resistance to intranasal inoculation. Whole-chicken-blood-agar plates were used as a cultural medium. With this medium the presence or absence of the streptococcus can be easily determined by the characteristic colony and the resulting hemolysis. The results are shown in table III.

Table III shows that at least under experimental conditions carriers of infection exist among survivors of intranasal inoculation which show no outward manifestation of the disease. Birds 23 and 27 are of considerable interest even though they finally succumbed to the infection. Such birds as these would be a constant source of potential infection to susceptible birds in a flock with which they were associated.

TABLE III—Results of nasal cultures made from birds showing resistance to the disease.

BIRD	DATE OF INOCULATION	SWABBING DATES	RESULT
6	4-12-29	4-20-29	+
		5-12-29	+
		5-24-29	+
		6-20-29	+
		7-6-29	+
		8-5-29	+
23	4-12-29	4-20-29	+
		5-12-29	+
		5-24-29	+
		6-20-29	+
			D. 5-24-29
24	4-19-29	5-12-29	+
		5-24-29	+
		6-20-29	—
		7-16-29	—
		8-5-29	—
26	4-19-29	5-12-29	+
		5-24-29	+
		6-20-29*	—
		7-16-29	+
		8-5-29	+
27	4-19-29	5-12-29	+
		5-24-29	+
		6-20-29	+
			D. 7-6-29

\*Spreader on plate.

Mr. R., the owner of the flock in which the disease occurred, was asked for permission to swab the survivors, but unfortunately this request was not granted. It was impossible, therefore, to demonstrate the carrier state in birds under field conditions.

#### LOCALIZATION

Two birds, (23 and 27, table I) are of interest in this connection. Bird 23 was given 0.2 cc of a 24-hour blood-broth culture intranasally on April 12. The following day this bird was accidentally selected by a laboratory assistant to supply blood for making whole-blood-agar plates. Some of the plates were incubated over night at 37° C. and on the following day

showed many hemolytic colonies. Since a measured amount of blood was used in each plate, it was possible to determine the number of organisms per cc contained in the blood at the time of bleeding. By actual plate count, therefore, it was found that the blood contained approximately 1,000 organisms per cc 24 hours after inoculation. Two days after inoculation a profuse stringy discharge was seen coming from both eyes. This condition persisted for several days. On the third day following inoculation, a subcutaneous edema was observed to involve the head parts. This increased in severity until the head of the bird appeared to be twice the normal size. The edema persisted for about two weeks and then subsided. After another two weeks the edema reappeared and was as severe as on the first occasion; it persisted for about two weeks. The bird died June 24, 72 days after inoculation. During this time the nasal cavity was cultured on four different occasions, and the organism recovered each time (table III). Autopsy of the bird revealed a large amount of caseous exudate involving the subcutaneous tissues about the head and the upper neck region. Cultures from the heart-blood remained sterile. However, the organism was recovered from the subcutaneous tissue around the head. It should be noted, however, that at this time the organism was not recovered in pure culture.

Bird 27 was inoculated intranasally April 19, and was very sick three days following inoculation. However, after two weeks it seemed to be much better, but never fully recovered. It died July 6, 78 days after inoculation. On autopsy the left lobe of the liver was found to be covered with a dry fibrinous exudate. This lobe was atrophied, dry and presented an undulated surface. The right lobe was normal. Cultures from the heart-blood remained sterile. The streptococcus, however, was isolated from the left lobe of the liver. The nasal cavity of this bird was cultured on three different occasions and the streptococcus recovered each time.

#### DISCUSSION

The data contained in this report show the existence among chickens of a highly fatal septicemia, due to a hemolytic streptococcus. The organism isolated in this outbreak appears to be not unlike the streptococcus producing a similar disease, described by Norgaard and Mohler. It would seem that this disease is of rather rare occurrence. However, the incidence cannot be estimated accurately, for no doubt a great many outbreaks of disease among poultry escape the attention of one able to make a definite diagnosis.

The experiments here presented show that the streptococcus is not capable of producing disease when introduced directly into the alimentary tract. However, when the organism is introduced into the nasal cavity, it gives rise not only to a septicemia but also to various types of localized infections. Still another form of the infection, which is not readily recognizable, is the healthy nasal carrier state. It appears also that some birds will resist infection entirely, even though they are given intranasally a dose of the organism which is known to be fatal to other birds similarly inoculated.

The fact that some birds, following intranasal inoculation, remain carriers of the infection, indicates that the disease becomes established in a flock through the introduction of such individuals. On the other hand, the converse of this may take place, in which susceptible birds are introduced into a flock in which carriers exist. The origin of the disease in the various outbreaks reported can be explained on the above basis. In the outbreak here reported, it is known that adult birds were introduced into the flock affected about two weeks before the disease appeared. Norgaard and Mohler made a similar observation, but they did not consider the birds introduced a factor in the origin of the disease. Greve noted that only Minorca pullets which had been reared from hatching eggs were affected, while the adult native birds with which they associated remained healthy.

Magnusson observed that the Single Comb White Leghorns and Barred Plymouth Rocks in the affected flock were quite susceptible to the disease, while the native races with which they associated resisted the infection.

The periodicity for the disease is indicated by the fact that all the outbreaks of the infection, that have been reported, have had their beginning in either October, November or December. The outbreak may be of short duration or may continue throughout the winter, but usually the disease disappears with the coming of warm weather.

#### SUMMARY

1. A specific infectious disease of chickens caused by a hemolytic streptococcus is described.
2. The causative organism occurs abundantly in the blood and organs in chains of 2 to 20 cells, with chains of 6 to 8 elements predominating.
3. The portal of entry is shown to be by way of the nasal cavity and not by way of the alimentary tract.

4. It is demonstrated that some birds resist intranasal inoculation but become carriers of the infection, and that the organism may be isolated from the nasal cavity of such individuals.

5. Localization of the infection following intranasal inoculation is shown in the case of two birds.

6. The disease probably becomes established in a flock through the introduction of carrier birds or by the introduction of susceptible birds into a flock in which carriers exist.

#### REFERENCES

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<sup>2</sup>Dammann, C., and Manegold, O.: Die Schlafkrankheit der Hühner. Deut. Tierärztl. Wchnschr., xiii (1905), pp. 577-579.  
<sup>3</sup>Greve, L.: Beitrag zur Kenntniss der Streptokokken-krankheit (Schlafkrankheit) der Hühner. Deut. Tierärztl. Wchnschr., xv (1908), pp. 213-215.  
<sup>4</sup>Magnusson, H.: Über eine für Europa neue Hühnerseuche. Apoptektische Septikämie der Hühner. Cent. f. Bakt., Abt. I, Orig. Bd. lvi (1910), pp. 411-428.

### Report from South Dakota

Dr. G. P. McCue, A. V. M. A. resident secretary for South Dakota, reports that eleven counties in his state voted on the question of abolishing the office of county agricultural agent at the fall election in 1932, and the proposal carried in nine counties. Two of the eleven counties voted to retain the county agent by small margins. This would indicate that only about thirteen of the 69 counties in South Dakota now employ county agents. Judging from reports, some of these have been rather troublesome to veterinarians. South Dakota is one of the states that conduct vaccination schools for farmers.

### Man Bites Cat

The old definition of news, "When a man bites a dog, it's news," was switched from the canine to the feline when a man in Raleigh, N. C., playfully (he thought) bit a cat's tail. Judge N. A. Sinclair (not playfully) sentenced him to 90 days.

### More Sows to Farrow in 1933

An estimate of the number of sows to farrow in the United States between December 1, 1932, and June 1, 1933, is set at 8,709,000 head, a two per cent increase over the corresponding period of the previous year.

"Do you know what the letters 'U. S.' on my horse blanket mean?"

"United States?"

"No. Unsafe."

## THE RELATION OF AGE, BREED, AND SPECIES TO SUSCEPTIBILITY TO TRANSMISSIBLE LEUCOSIS OF CHICKENS\*

By E. L. STUBBS, *Philadelphia, Pa.*

*The Henry Phipps Institute and the Veterinary School  
University of Pennsylvania*

There are no systematic investigations known on the relation of age, breed and species to susceptibility to transmissible leucosis of fowls. Exact data on this subject are essential to show the best conditions under which leucosis of chickens may be investigated experimentally, and to throw light on the spread of the spontaneous disease and on the nature of the transmitting agent. For example, tumors and leucosis of mammals can be transmitted in several generations only in the species in which they originated. Successful heterotransfer, on the other hand, is characteristic for most virus diseases. Furthermore, the presence or absence of the agent transmitting leucosis is obviously best determined by injecting birds at an age when they are most susceptible to the disease.

### The Susceptibility of Fowls to Transmissible Leucosis at Various Ages

The experimental transmission of fowl leucosis has usually been carried out with young chickens. Whether old fowls are susceptible, and the whole question of the relation of age to susceptibility, have not hitherto been investigated.

Ellerman<sup>1</sup> says that young fowls are most suitable for inoculation. It is often maintained that tumors are most readily transmitted to younger animals. Rous<sup>2</sup> observed that young chickens are more susceptible to sarcoma than adults. It has been found at the Henry Phipps Institute that mice of any age are susceptible to transmissible lymphoid leucosis. Spontaneous lymphoid leucosis (so-called "big liver" disease) is rather common in older chickens, but we do not know that it has been reported in very young chickens.

A study of the relation of age to transmissible leucosis was undertaken with the hope that it would determine the age of chicken most suitable for experimental work and would explain failure of some investigators to transmit leucosis. The work has been divided into two experiments.

\*This investigation has been supported by a Fund for the Study of Leukemia and Related Diseases. Received for publication, May 25, 1932.

*Experiment 1:* Three groups of Barred Plymouth Rock chickens were used, the breed with which most of the work with transmitted leucosis has been done. The birds in one group were four months of age; in one group, one year old; and in the third, over two years old. All the chickens were kept under identical conditions. Each was injected intravenously with 0.6 cc of blood from a leucotic fowl.

In group 1, consisting of six 4-month-old chickens, weighing from 650 to 1,050 grams each at the time of injection, there were four successful inoculations. The incubating periods were 15, 22, 22 and 29 days, with an average of 22 days. The incubation period is regarded as the time elapsing between injection and the first blood changes noted in blood-smears. Blood-smears were taken each week, so that some incubation periods actually may have been shorter than those recorded. The leucosis produced in the four fowls progressed to a fatal termination, indicating highly virulent inoculating material and marked susceptibility.

In group 2, six 1-year-old chickens, weighing from 1,230 to 1,950 grams each when injected, there were likewise four successful inoculations. The incubation periods were 15, 36, 54 and 79 days, with an average of 46 days. The chicken that showed the first blood changes in 15 days developed the disease progressively and died three weeks later. The chicken with an incubation period of 54 days showed at first the blood changes of erythroleucosis and mild myeloid leucosis. One month later this chicken had a count of 850,000 red blood-cells and 135,000 white blood-cells. Severe leucosis continued for another month and the chicken died 64 days after the first blood changes were detected and 118 days after being injected. On postmortem examination the liver weighed 60 grams and the spleen 20 grams. The chicken with the incubation period of 36 days showed blood changes for about one month, after which it recovered and when killed, about four months later, showed nothing to indicate leucosis. Similarly the fowl with an incubation period of 79 days showed blood changes characteristic of erythroleucosis and myeloid leucosis for six weeks and then was negative. It was killed about two months after the last blood changes observed and at this time there was no indication of leucosis.

Group 3 included six old Barred Plymouth Rock roosters and hens of unknown exact age but more than two years old. There were two successful inoculations. The incubation periods were 43 and 53 days. The disease developing after 53 days continued to a fatal termination three days later; at postmortem examination the liver weighed 160 grams and the spleen 25 grams. The fowl with the 43-day incubation period showed no further blood alteration ten days later and, when killed four months later, showed nothing to indicate leucosis.

This experiment showed that chickens in each of three age periods acquired leucosis. Inoculation was successful in 66 $\frac{2}{3}$  per cent of the 4-month-old and the 1-year-old chickens, and in 33 $\frac{1}{3}$  per cent of those more than two years old. There was an average incubation period of 22 days, with a minimum of 15 days and a maximum of 29 days, in the four transmitted cases in the youngest group; an average of 46 days, with a minimum of 15 days and a maximum of 79 days, in the four cases in the one-year-old group; an average of 48 days, with a minimum of 43 days and a maximum of 53 days, in the two cases in the

oldest group. This suggests that the younger the chicken the more marked the susceptibility as evidenced by the shorter period of incubation and the increased percentage of successful inoculations. There were four deaths in the 4-month-old group, two in the 1-year-old group, and one in the older group, suggesting that the younger the chicken the more fatal the disease. (See table I.)

TABLE I—Results of transmission experiments, using fowls of different ages.

GROUP	APPROXIMATE AGE	FOWLS INJECTED	SUCCESSFUL	UNSUCCESSFUL	AVERAGE PERIOD OF INCUBATION	RECOVERED	DIED
1	4 months	6	4	2	22	0	4
2	1 year	6	4	2	46	2	2
3	2 years or older	6	2	4	48	1	1

*Experiment 2:* Opportunity was presented also to test further the transmissibility of fowl leucosis in various ages by including baby chicks and some old fowls. Table II is a summary of the results.

TABLE II—Results of transmission experiments, using baby chicks and older fowls.

GROUP	APPROXIMATE AGE	FOWLS INJECTED	SUCCESSFUL	UNSUCCESSFUL	AVERAGE PERIOD OF INCUBATION	RECOVERED	DIED
1	4 days	3	3	0	19	0	3
2	18 days	5	3	2	49	0	3
3	23 days	7	6	1	23	0	6
4	2 months	4	3	1	20	1	2
5	2 to 4 years	6	2	4	45	0	2
6	Over 3 years*	3	0	3			

\*Age not definitely known.

Group 1 consisted of three chicks that were hatched in the Institute. When four days old, each was injected intravenously with 0.01 cc of leucotic blood. At the same time, four controls, Barred Plymouth Rocks four months old, were injected intravenously each with 0.2 cc of the same leucotic blood. All four died of leucosis after incubation periods of 15, 17, 21 and 28 days and after a sickness of from 3 to 31 days. All three of the baby chicks died with leucosis after incubation periods of 15, 21 and 21 days and a sickness of 9, 12 and 51 days. The average incubation period (19 days), was the shortest in any group so far observed.

Group 2 included five White Leghorn chicks eighteen days old inoculated with 0.1 cc of leucotic blood. Two died of leucosis, one 122 and one 96 days after inoculation, after a sickness of 15 and 55 days, re-

spectively. The three other chicks died 19, 20 and 24 days after injection; of these the one dying the 20th day alone showed microscopic lesions characteristic of leucosis.

Group 3 included seven chicks 23 days old, each injected with 0.1 cc of leucotic blood. All but one developed leucosis after incubation periods of from 16 to 26 days, the average being 23 days, and all six died. Four Barred Plymouth Rock chickens were used as controls, each weighing about 750 grams and each injected with 0.5 cc of the leucotic blood. Three of the controls developed leucosis after an incubation period of 35 to 42 days; the fourth had a severe transient anemia but when killed did not show any leucotic changes on post-mortem examination.

Group 4 consisted of four 2-month-old chickens injected with leucotic blood. Three developed leucosis from 14 to 30 days after injection, the average incubation period being twenty days. Two died. The fourth chicken developed a transient anemia.

Group 5 included six hens\* from two to four years old injected 49 days previously with blood that had proved avirulent. Each was re-injected with 0.5 cc of blood from a leucotic chicken. Two developed leucosis 45 days after injection. Four remained healthy and of these, three were again injected with leucotic material but remained free of leucosis even five months after the third injection. Three control fowls weighing about 800 grams were injected with the same leucotic blood at the same time but only one developed leucosis.

Group 6 included three chickens presumably over three years of age. Each was injected with 1 cc of leucotic blood from the donor used for group 3. None of these old chickens showed any indication of leucosis in blood smears or on postmortem examination performed 137 days after injection.

Experiment 2† shows again that young chicks are more susceptible to leucosis and that leucosis is more fatal in them. The source of leucotic blood in this experiment was not the same as in experiment 1, and consequently there may have been some variation in the infectivity of injected material. This experiment included old fowls of known age.

It will be noted from table II that inoculation was successful in 100 per cent of group 1, consisting of three 4-day-old chicks, with an average incubation period of 19 days and a 100 per cent fatality. This is the shortest period of incubation and the highest mortality in any group so far observed.

In group 2 three of the chicks inoculated at the age of 18 days died earlier than is usually required for the development of leucosis. On the other hand the two that survived had longer than usual periods of incubation. Intercurrent disease probably interferes in some cases with the development and progress of transmissible leucosis. It is possible, however, that the injected

\*We wish to thank members of the staff of the Pennsylvania State College Department of Poultry Husbandry for sending us the chickens included in group 5.

†The author acknowledges with thanks the assistance in this work of members of the staff of The Henry Phipps Institute.

material given this group may not have been so virulent as that given other groups.

Group 3 showed a high incidence of disease and a high mortality, indicating marked susceptibility in these chicks, inoculated at the age of 23 days. None of the old chickens in group 6 developed leucosis. It may be that the material used was not of high enough virulence to produce the disease. Group 5, however, consisting of chickens of known ages (from 2 to 4 years) were injected with the same material. Two acquired leucosis and died. This shows that old chickens may become affected.

The work of this experiment confirms previous observations<sup>2</sup> showing that all ages seem to be susceptible but that older birds are less susceptible to transmissible leucosis than younger birds.

Since the above work has been completed, Engelbroth<sup>4</sup> has reported a transmissible strain of erythroleucosis that at first could be transmitted to chickens of all ages but later only to baby chicks.

#### SUMMARY

It is shown that baby chicks, older chicks, young chickens and old chickens may become affected with transmissible leucosis.

Baby chicks four days old showed 100 per cent incidence of disease after inoculation with an average incubation period of 19 days and 100 per cent mortality. Old chickens showed a 37 $\frac{1}{2}$  per cent incidence and a 75 per cent mortality.

It is concluded that chickens of all ages may become affected with leucosis. The younger the chicken the higher the susceptibility as measured by the period of incubation, the percentage of successful inoculations, and the mortality.

#### The Susceptibility to Transmissible Leucosis of Various Breeds of the Domestic Fowl

Accurate information regarding the susceptibility of various breeds of domestic fowl to leucosis is wanting. The statements in the literature on the subject have to be revised to take into consideration that lymphoid leucosis is separate from myeloid leucosis and erythroleucosis. Little is known of the former disease; the latter are caused by a filtrable agent. A study of susceptibility to transmissible leucosis has been made with a strain isolated in 1929.<sup>5</sup>

Six groups of six members each of various breeds of chickens including Barred Plymouth Rocks, White Leghorns, Rhode Island Reds, Bantams, Naked Necks and mixed breeds were inoculated each with 0.5 cc of whole leucemic blood of fowls with severe transmissible leucosis. Table III records the results.

TABLE III—Results of transmission experiments, using various breeds of chickens.

GROUP	BREED	FOWLS IN- JECTED	SUC- CESSFUL	UNSUC- CESSFUL	AVERAGE PERIOD OF INCUBATION	RE- COV- ERED	DIED
1	Barred Rocks	6	4	2	22	2	2
2	White Leghorns	6	4	2	43	2	2
3	Rhode Island Reds	6	2	4	29	1	1
4	Bantams	6	6	0	16	1	5
5	Naked Necks	6	3	3	22	0	3
6	Mixed breeds	6	2	4	22	1	1

Group 1, consisting of six Barred Plymouth Rock chickens, weighed 650 to 1,050 grams at the beginning of the experiment. There were four successful inoculations, as shown by blood smears, after an average incubation period of 22 days. These chickens were pale and lost weight while affected with leucosis. Two died after a sickness of 12 and 24 days, respectively. Two recovered after a sickness of 10 and 21 days, respectively.

Group 2, consisting of six White Leghorn chickens, weighed 675 to 960 grams at the start of the experiment. There were four successful inoculations as evidenced by blood-smears after an average incubation period of 43 days. The average incubation period in this group was considerably longer than that of any of the other groups. This was due to the fact that one chicken had an incubation period of 54 days and one a period of 74 days. The other two successful injections had incubation periods of 23 days each. The chicken with an incubation period of 54 days suffered a long while with leucosis and died after a sickness of 107 days. At this time its carcass weighed only 800 grams, having decreased from a previous weight of 1,200 grams. This chicken at postmortem examination showed swollen bone-marrow, a swollen liver, weighing 80 grams, and a swollen spleen weighing 20 grams. The one with an incubation period of 74 days continued with blood alterations of the erythroleucosis type for three months, after which no blood alteration was found. This chicken was killed two months later, that is, five months after the first blood change was noted, and at postmortem examination no gross changes were noted. One of the chickens with a 23-day incubation period recovered while the other died 17 days later, showing gross changes of leucosis. This chicken at death weighed 855 grams and had a liver weighing 50 grams and a swollen spleen weighing 10 grams.

These Leghorns were not in such good physical condition as, especially, those of groups 1 and 4. One of them died from air-sac infection on the 23rd day after injection and another one died from air-sac infection and pneumonia 83 days after injection. This chicken showed large numbers of polymorphonuclear leucocytes in the blood before death. We believe that this intercurrent disease was present when the chickens were received and was not due to the injection. It is probable that the presence of intercurrent disease interferes in some cases with the development and progress of transmissible leucosis.

Group 3, consisting of six Rhode Island Red chickens, weighed 970 to 1,210 grams at the start of the experiment. Leucosis developed in two of these birds; in one after 22 days, in the other after 36 days,

the average incubation period being 29 days. The one developing leucosis first recovered in about one month and when killed four months later showed no evidence of leucosis on postmortem examination. In the other the disease progressed until there were cells of both erythroleucosis type and myeloid leucosis type in the blood. The weight of this chicken was 1,210 grams at the time of injection, the heaviest fowl in any of the groups. Forty-two days after injection and six days after the first blood changes were found, it weighed 1,670 grams. It died after showing blood changes for 20 days. At death the carcass weighed 1,250 grams, the enlarged liver weighed 200 grams, and the spleen 50 grams.

Group 4 consisted of six Bantams weighing from 570 to 770 grams each at the beginning of the experiment. All of the Bantams became sick with leucosis after an average incubation period of 16 days. The blood alterations were noted first at 14 days in four and at 21 days in the two others. Four of these Bantams died after sickness lasting from 21 to 48 days. One died after a sickness of 106 days and showed blood alterations during the entire time. One apparently recovered and after a sickness of 23 days showed no further blood alteration. This Bantam was killed 140 days after the time that the first blood changes were noted and on postmortem examination no gross changes of leucosis were found.

Four control young Barred Rock chickens were injected with blood from the same donor used for the Bantams and of these there were three successful inoculations. These fowls, like the Bantams, had a minimum incubation period of 14 days and a maximum of 21 days. Two died after sicknesses of 23 and 30 days, respectively. The third died one day after the first blood changes were noted and at postmortem examination showed no gross changes to indicate leucosis.

Group 5 consisted of six Naked Necks or turkens. These weighed from 1,800 to 2,800 grams at the beginning of the experiment. Each was injected intravenously with 2.5 cc of leucotic blood. At the same time three young Barred Plymouth Rock chickens were injected intravenously for control purposes each with 2 cc of the same leucotic blood. These weighed about 800 grams each. One developed leucosis after a period of 60 days and died 16 days later, showing leucosis. Another developed a transient leucosis in 28 days and recovered after a period of ten days. The third control chicken was negative.

Three Naked Necks developed leucosis after incubation periods of 19, 21 and 28 days, with an average of 22 days. All three died after sicknesses of 14, 15 and 22 days. The infecting material in this instance did not seem particularly active as measured by its effect on the control chickens, yet half of the injected Naked Necks developed leucosis, showing their susceptibility.

Group 6 included six mongrel chickens of mixed breeds and colors, weighing 650 to 1,090 grams at the beginning of the experiment. Each was injected with 0.6 cc of heparinized whole blood of chicken 1182, which was affected with leucosis. This was the same donor from which leucotic blood was obtained to inject the first three groups. There were two successful inoculations, as evidenced by blood changes first noted in both on the 22nd day. One of these progressed to a mixed type of disease showing both erythroleucosis and myeloid leucosis and died after a sickness of 25 days. The other one recovered in about one month and, when killed one month later, showed no changes of a leucotic nature on postmortem examination.

### BLOOD RELATIONSHIP BETWEEN TURKEN, TURKEY AND THE COMMON FOWL

At the time this experiment was undertaken there was current a belief that the Naked Neck or turken is a cross between the turkey and the chicken. Blood serum from these birds was injected into chickens and turkeys and vice versa to see if precipitins could be produced. These inoculations did not result in the production of precipitins in chickens against turken serum or in turkens against chicken serum, whereas both produced precipitins when injected into turkeys. This indicates that turkens or Naked Necks are a breed of chickens and have no blood relationship to turkeys.

#### SUMMARY

A study has been made of the susceptibility to transmissible fowl leucosis of various breeds of chickens, in six groups of six fowls each, which shows the susceptibility of all breeds as previously reported by Stubbs and Furth.<sup>3</sup> Inoculation was successful in 33⅓ per cent of the Rhode Island Reds and the mixed breeds, in 50 per cent of the Naked Necks, in 66⅔ per cent of the Barred Plymouth Rocks and the White Leghorns, and in 100 per cent of the Bantams. The experiment shows that some members of each of six groups of different breeds of chickens are susceptible to transmissible leucosis and suggests that all breeds of chickens are similarly susceptible. Precipitin tests indicate that the turken or Naked Neck is a breed of chicken.

### Attempts to Transmit Leucosis from Chickens to Other Species of Fowl

It has been shown above that chickens of all ages and of various breeds may be affected with transmissible leucosis of the domestic fowl. The question naturally arises whether fowls other than chickens are susceptible. Observations showing that the etiological agent is present in the blood and is filtrable suggest that it has a virus-like nature.<sup>6</sup> Other diseases of fowls caused by filtrable viruses, such as fowl-pox and fowl-pest, are transmissible to several species. Successful transfer through several subsequent passages from chickens to other kinds of fowl would favor the view that the transmissible agent of avian leucosis is an ordinary virus. It is noteworthy that the Rous tumors of chickens until recently were not transmitted to other species. Fujinami's myxosarcoma, however, has been transmitted to ducklings and very recently Des Ligneris<sup>7</sup> has succeeded in trans-

planting Rous tumors in guinea fowls and turkeys and Andrewes<sup>8</sup> in pheasants.

The susceptibility of guinea fowls, pigeons, turkeys, ducks, geese, pheasants, and one hybrid fowl to leucotic blood of chickens has been tested. Table IV is a summary of the results.

TABLE IV—Results of transmission experiments, using various species of fowl.

EX- PERI- MENT	SPECIES	FOWLS IN- JECTED	SUC- CESSFUL	DIED OF INTERCURRENT DISEASE	CONTROL CHICKENS	
					IN- JECTED	DEVELOPED LEUCOSIS
1	Guinea	10	0	0	0	0
2	Guinea	10*	0	0	4	2
3	Pigeon	6	0	0	12	8
4	Pigeon	6*	0	0	4	2
5	Pigeon	6	0	0	6	6
6	Turkey	5	0	3	3	0
7	Turkey	5	0	1	4	3
8	Turkey	2*	0	0	4	4
9	Turkey	4	0	0	2	2
10	Duck	6	0	0	4	3
11	Goose	4	0	0	4	0
12	Goose	6	0	0	4	3
13	Goose	4*	0	0	4	4
14	Pheasant	5	0	3	4	4
15	Pheasant—Ban- tam hybrid	1	1	1		

\* Re-injected.

#### ATTEMPTS TO TRANSMIT LEUCOSIS TO GUINEA FOWLS

*Experiment 1:* Ten guinea fowls several months old were injected intravenously with amounts of leucotic blood varying from 0.5 to 2 cc. Two chickens were donors in this experiment. Following injection none developed leucosis as manifested by signs of physical disturbance or in blood examinations made every two weeks over a period of four months.

*Experiment 2:* The same 10 guinea fowls four months after the first inoculation were injected again, each with 0.5 cc of leucotic blood. Four control chickens also were injected with 0.5 cc of leucotic blood from the same donor, and two (50 per cent) developed leucosis. Again no symptoms were produced in the guinea fowls and blood-smears, made every two weeks for another four months, showed no indication of leucosis.

#### ATTEMPTS TO TRANSMIT LEUCOSIS TO PIGEONS

*Experiment 3:* Six pigeons were injected intravenously each with 0.5 cc of leucotic blood. At the same time twelve young chickens also were injected intravenously with 0.5 cc of leucotic blood from the same donor, and eight (75 per cent) developed leucosis. None of the pigeons were abnormally affected and periodic blood examinations were always negative for leucosis.

*Experiment 4:* The same six pigeons four months later were all re-injected with 0.5 cc of blood from another leucotic chicken. At the same time four young control chickens were injected intravenously each with 0.5 cc of the same leucotic blood, and two (50 per cent) developed leucosis. Again no disturbance of any kind was produced in the pigeons.

*Experiment 5:* Each of six young pigeons was injected with 0.5 cc of leucotic blood. Six Bantam chickens also were injected intravenously with 0.5 cc of leucotic blood from the same donor. All six controls developed leucosis, indicating a high degree of infectiousness for this material. Again no disturbance of any kind was produced in the pigeons.

#### ATTEMPTS TO TRANSMIT LEUCOSIS TO TURKEYS

*Experiment 6:* Five young turkeys about three weeks old were injected with 0.5 cc of leucotic blood and at the same time three chickens were injected with blood from the same donor. None of the control chickens developed leucosis and none of the turkeys developed leucosis. Three of the turkeys died of intercurrent disease.

*Experiment 7:* Five young turkeys about three weeks old were injected with 0.5 cc of leucotic blood. Four control chickens were injected intravenously with 0.5 cc of blood from the same donor and three developed leucosis, one after an incubation period of 16 days and two after a period of 33 days. None of the turkeys inoculated with this highly infectious material developed leucosis. One died of intercurrent disease on the 17th day following injection.

*Experiment 8:* Two turkeys of experiment 6 that lived to maturity were re-injected with leucotic blood. All of four control chickens similarly injected developed leucosis. Neither of the turkeys developed leucosis. Later these two turkeys were re-injected several times with leucotic blood but did not develop leucosis.

*Experiment 9:* Four turkeys, two small ones weighing 770 and 940 grams, and two large ones weighing 3,980 and 3,720 grams, were injected with from 2 to 10 cc of leucotic blood. This blood was obtained from a Naked Neck chicken. At the same time two chickens were injected with blood from the same Naked Neck and both within 12 days developed leucosis, which was fatal after a sickness of one week. None of the turkeys developed leucosis from this highly active leucotic blood.

Forty-eight days after the first injection, these four turkeys were re-injected with leucotic blood again without success.

#### ATTEMPTS TO TRANSMIT LEUCOSIS TO DUCKS

*Experiment 10:* Six young ducks about three weeks old were injected with 0.5 cc of leucotic blood. Three of four control chickens injected intravenously with 0.5 cc of the same blood developed leucosis. None of the ducks showed any indication of leucosis in repeated blood-smears or by any disturbance in their health or growth.

#### ATTEMPTS TO TRANSMIT LEUCOSIS TO GEESE

*Experiment 11:* Each of four young geese about three weeks old was injected intravenously with 0.5 cc of leucotic blood. Four chickens were injected with blood from the same donor. None of the controls developed leucosis, nor did any of the geese develop the disease.

*Experiment 12:* Six young geese about three weeks old were injected intravenously with 0.5 cc of blood. Blood from the same leucotic chicken was injected into four control chickens at the same time, and three developed leucosis, one after an incubation period of 16 days, and two after 33 days. None of the geese developed leucosis, as indicated by repeated blood-smears or by any interference with normal health and growth.

*Experiment 13:* The four geese used in experiment 11 were re-injected with 0.5 cc of leucotic blood two months following the first injection. All of four control chickens injected with blood from the same leucotic chicken developed leucosis in from 14 to 29 days, with

an average incubation period of 23 days. None of the re-injected geese developed leucosis.

#### ATTEMPTS TO TRANSMIT LEUCOSIS TO PHEASANTS

*Experiment 14:* Each of five young pheasants a few weeks old was injected with 0.25 cc of leucotic blood. All of four control chickens, weighing 750 grams, injected intravenously with 0.5 cc of the same blood, developed leucosis 15, 16, 18 and 23 days after injection and all died after sicknesses of from 1 to 26 days. Three of the five pheasants were pecked badly and died of injuries 24 days after injection. One died 44 days after injection and one remained healthy. Blood-smears and postmortem appearance of these pheasants showed nothing to indicate leucosis.

#### TRANSMISSION OF LEUCOSIS TO A HYBRID CHICKEN

*Experiment 15:* A hybrid, a cross between a pheasant male and a Bantam female, was obtained through the courtesy of Dr. F. R. Beaudette, of New Brunswick, N. J. After injection with leucotic blood, this bird developed leucosis and died after a sickness of 84 days. Three days after the first blood changes were observed in this leucotic hybrid its blood was injected into both chickens and pheasants. Some of the chickens developed leucosis, but none of the five pheasants showed leucotic symptoms or blood changes.

The last experiments were performed with the purpose of determining whether the transmitting agent shows signs of adaptability, a fundamental characteristic of life. It did not exhibit this property under the conditions described.

#### SUMMARY

Transmissible avian leucosis could not be reproduced by inoculation of leucotic blood of chickens into fowls of other species, namely, guinea fowls, turkeys, pheasants, pigeons, ducks and geese. A pheasant-chicken hybrid was successfully inoculated and from this leucemic hybrid the disease was passed readily to chickens but not to pheasants.

This work confirms previous studies reported by Stubbs and Furth<sup>3</sup> on the non-susceptibility of species other than chickens.

#### CONCLUSIONS

Transmissible leucosis of chickens can be transmitted readily to various breeds of chickens. It can be transmitted to chickens of various ages; the younger the fowl, the more susceptible. It can not be transmitted from chickens to birds of other species.

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## STAPHYLOCOCCAL ARTHRITIS IN TURKEYS\*

By ERWIN JUNGHER, *Storrs, Connecticut*

*Department of Animal Diseases  
Storrs Agricultural Experiment Station*

The syndrome of paralysis in gallinaceous birds apparently may be brought about by a variety of disease conditions, such as neuro-lymphomatosis, coccidiosis and intestinal parasitism. In view of the complexity of the problem, every case of paralysis subjected to etiological analysis is of value in the ultimate recognition of the underlying disease factors. During the past few years, several cases of lameness in young turkeys have been seen during the routine diagnostic work at the Storrs Station; some of them were obviously of rachitic origin, others resembled the so-called true fowl paralysis in their clinical features, without presenting the lesions of neuro-lymphomatosis gallinarum. Recently an outbreak of paralysis in young turkeys was observed that was later recognized as one of staphylococcal arthritis. Since the condition, in the opinion of the flock-owner, who was a physician, was thought to resemble perosis in chickens, as described by Titus and Ginn,<sup>1,2</sup> and since staphylococcal arthritis in turkeys has not been reported in the literature, as far as the present author is aware, a study of this case is presented for publication.

### LITERATURE

In 1871, Prah,<sup>3</sup> in Prussia, reported outbreaks of paralysis among young geese that were affected with severe arthritis of the leg and wing joints and succumbed within 2 to 3 weeks. In 1892, Lucet<sup>4</sup> described an infectious osteo-arthritis of young geese in France and was the first observer to incriminate *Staphylococcus aureus* as the etiological agent of the malady. During the first decade of the present century, the disorder again was recognized in Germany by Freese<sup>5</sup> and Loeffler<sup>6</sup> in geese and ducks. Freese observed an acute and a chronic form of the disease; in the former, which was usually fatal in 2 to 4 days, the birds suffered from severe diarrhea, general debility and painful swellings of the joints; in the latter the systemic symptoms were less pronounced, but the birds became gradually emaciated in spite of good appetite. The joints were markedly enlarged. Some chronic cases apparently recovered, but were subject to relapses. Anatomically, the condition was characterized by sero-fibrinous

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arthritis, hemorrhagic osteomyelitis, and suppurative ostitis. The author regarded *Staph. aureus* as the etiological agent, on the basis of cultural tests and experimental reproduction of the disease. Van Heelsbergen<sup>7</sup> observed the disease in Holland, and Hasenkamp and, Sachweh,<sup>8</sup> Stroh,<sup>9</sup> Eber<sup>10</sup> and Reinhardt<sup>11</sup> in various provinces of western Germany. Hutyra and Marek<sup>12</sup> described the disease under the caption of "staphylomycosis," Kaupp<sup>13</sup> as "osteo-arthritis," and Buckley, Bunyea and Cram<sup>14</sup> as "lameness in geese and ducks."

While the malady in the species named appears to be an established disease entity, its occurrence in chickens is rare or not fully authenticated, according to van Heelsbergen.<sup>15</sup> In his discussion of staphylococcosis in fowl, he quoted a report by Kraus on chicken losses attributed to *Staph. aureus* infection. In view of the symptomatology and rapid course of the disease, van Heelsbergen thought the etiological significance of the organism isolated was not established. Eber,<sup>16</sup> however, observed an outbreak of polyarthritis in young chickens that corresponded in all respects to the disease in geese and ducks.

Recently Hole and Purchase,<sup>17</sup> in England, described an outbreak of arthritis and periostitis in pheasants caused by *Staph. aureus*. In one instance they isolated *Staphylococcus citreus*. Birds from 6 to 10 weeks old became affected, showing lameness, hobbling gait, and resting on the hock joints. Anatomically the authors found the joints, especially the tibiotarsal ones, swollen by cheesy membranous deposits located either in the articular cavity or in the tendon sheaths; occasionally the pericardium was affected. The disease occurred in an acute septicemic and in a chronic arthritic form. The etiological agent was isolated from the heart-blood in the acute cases, and from the affected joints where the disturbance was chronic. It was definitely pathogenic to young birds, but the reproduction of the chronic arthritic forms could be effected in only a few cases. The authors thought wound infections possible, due to thistles as a predisposing factor, and found vaccines ineffective in control work.

#### AUTHOR'S OBSERVATIONS

During January, 1932, four birds from a large Bronze turkey farm in the State were submitted for diagnosis. The owner stated that, in a lot of about 450 seven-month-old turkeys, 25 had become lame and remained so for periods varying from three weeks to two months. Some of the affected birds showed im-

provement on removal to a barn; others became very emaciated and died.

Clinically, the poults presented well-developed specimens with strong bones, but somewhat droopy and emaciated. The paralytic symptoms varied so much as to make the etiological unity of the condition appear doubtful. While two of the birds were still able to support themselves on their legs, they could move backward only with a peculiar hopping motion, showing all the evidences of severe incoördination and ataxia of the locomotor apparatus. One poult kept one leg extended and turned backward, apparently affected by a flaccid paralysis; the other bird rested



FIG. 1. Leg of turkey, arthritis of hock joint (x 1/5).

on its hock joints most of the time, or moved only with great difficulty.

Pathological examination of the birds was negative, except for the joints. In all four cases the tibiometatarsal joints were enlarged and fluctuating. They appeared to be hot and painful, and were affected with superficial ulcerations comparable to decubital sores (fig. 1). The swellings in the metatarsophalangeal joints were slight and, if present alone, would be ordinarily

diagnosed as "bumblefoot." The femorotibial joints did not present any outward lesions, except a certain degree of puffiness. On sectioning of the tibiotarsal joints, it was apparent that the integument was thickened markedly and separated from the articular ligaments by serous exudate. Probably the outstanding and most characteristic lesion was found in the fibrinous deposits on the subcutis and the periarticular ligaments, here and there interspersed with hemorrhagic areas (fig. 2). The exudative processes extended distal and proximal of the articular region, as indicated by edematous changes along the tendinous sheaths and, in the case of the patellar joints, along the muscle fasciae.

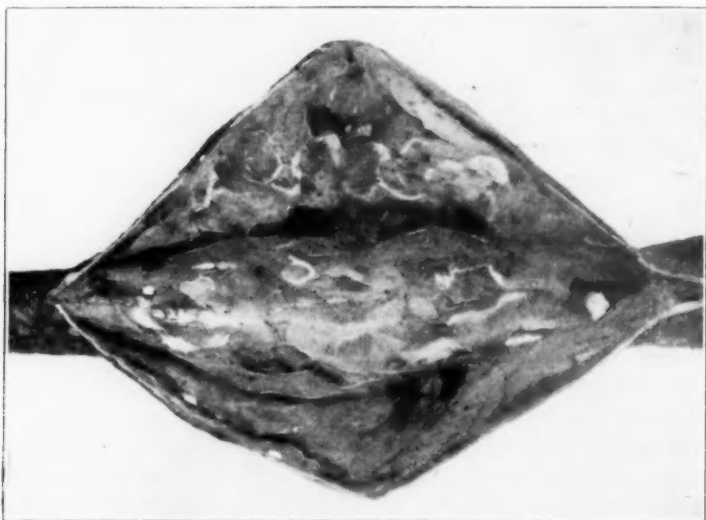


FIG. 2. Hock joint of turkey, volar subcutaneous space exposed; massive fibrinous deposits.

In some birds in which the swollen joints appeared rather firm to the touch, the enlargement was brought about by layers of dry, yellow, cheeselike material sandwiched between gelatinous tissue. The articular surfaces proper appeared to be normal, but the synovial bursa was enlarged and hemorrhagic.

In histopathological sections of the central nervous system, a search was made for lymphocytic perivascular cuffs, with negative results. Some small areas of the lumbar spinal column showed local erythrocytic extravasations which were interpreted as regional compression hemorrhages. The histological picture of the affected joints was confirmatory of the gross lesions, in that fibrinous deposits prevailed in the subcutaneous space. The

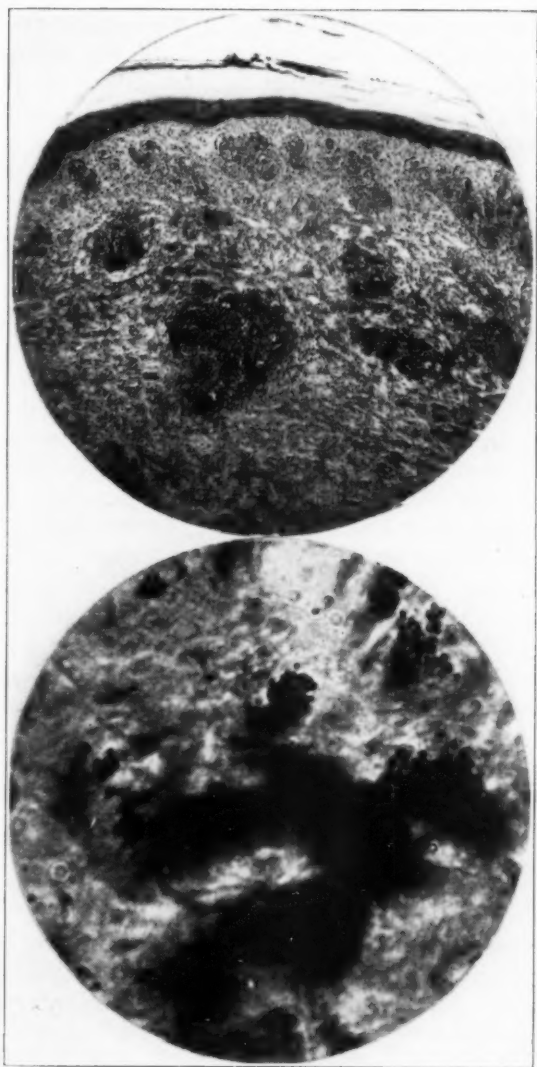


FIG. 3 (above). Section of hock joint; perivascular infiltration with pseudoeosinophiles (x 100).

FIG. 4 (below). Section of hock joint; the black areas are masses of cocci; the detailed morphology of the organism can be seen in the right upper quadrant (x 1800).

tissue elements appeared to be separated in evidence of the extant inflammatory edema. Close to the periphery the blood-vessels (fig. 3) were thickened markedly and compressed by pseudo-eosinophile perivascular infiltrations. In the deeper regions the gross architecture of the pus layers was carried out in microscopic detail in that dense aggregations of pseudo-eosinophiles changed off with layers of edematous slightly infiltrated tissue.

Bacteriological examination of the subcutaneous areas, synovial fluid and synovial bursa yielded an organism belonging to the Coccaceae. Cultures from the internal organs were negative. It was interesting to note that the organism could be isolated from joints of the posterior extremities which did not show any marked gross lesions as, for example, the knee and the hip joint, a fact that would point to the distribution of the arthritic infection in the body and exclude the possibility of dealing with an extraneous contaminant. The organism was a Gram-positive coccus, 0.8 to 1 $\mu$  in diameter and arranged in grape-like clusters. It grew on ordinary laboratory media, forming opaque, convex, spherical colonies of golden-yellow color. It acidified and coagulated litmus milk in 24 hours, produced sacciform liquefaction in gelatin stab cultures, did not produce indol, and reduced nitrates to nitrites.

The organism produced acid without gas in dextrose, galactose, glycerin, lactose, levulose, maltose, mannite, mannose and sucrose; it did not attack arabinose, dextrin, dulcitol, inulin, raffinose, salicin or xylose. In so far as the carbohydrate reactions are listed by Hole and Purchase,<sup>17</sup> the organisms isolated from turkeys and pheasants gave similar biochemical reactions, except in dextrin; however, dextrin is usually considered unreliable as a differential sugar. In toto, its characters correspond to the accepted description of *Staph. aureus*.

The etiological significance of the organism in the disease was inferred from the complete agreement of the symptomatologic, pathologic and bacteriologic features of the disorder in turkeys with those described in geese, ducks and pheasants. Furthermore, it was possible to demonstrate the causative agent as masses of Gram-positive cocci in histological sections of the affected joints (fig. 4).

#### SUMMARY

An infectious disease characterized by polyarthritic symptoms and more or less of a systemic reaction is known to occur in geese and ducks; sometimes it affects young chickens and pheas-

ants. The disease is caused by *Staphylococcus aureus* (occasionally *Staphylococcus citreus* has been observed) and is described in the literature as staphylococcosis, staphylomycosis, lameness of geese and ducks, osteo-arthritis, etc.

In including staphylococcus infections of gallinaceous birds in the disease entity under discussion, two criteria must be taken into consideration: lameness and arthritic affections.

Staphylococcal arthritis in turkeys has been observed in Connecticut; the occurrence of the malady in turkeys is apparently reported here for the first time.

According to observations of other authors, the disease occurs in an acute septicemic and in a chronic arthritic form. Only the latter has been observed in turkeys, but the former may occur and should be watched for by the diagnostician.

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### Frozen Eggs Seized

The biggest seizure of canned frozen eggs in the history of enforcement of the federal Food and Drugs Act was effected recently, when the government embargoed more than 23,000 thirty-pound cans, valued at approximately \$100,000, according to federal officials. The 20 carloads of eggs were partially decomposed, therefore, adulterated under the national pure food and drug law.

Officials of the federal Food and Drug Administration frequently in the past have been obliged to seize frozen eggs, on the ground of partial decomposition. The Food and Drugs Act specifically classes a food as being adulterated if it shows evidence of decomposition or if it contains added deleterious, harmful, or unwholesome material.



## CRANIAL NERVE PARALYSIS\*

By L. R. BARTO, *Summit, N. J.*  
*J. B. Engle Veterinary Hospital*

### INTRODUCTION

Following are the reports of two very unusual cases treated at this hospital. They were entered under the tentative diagnosis—suspicious of rabies. The latter dread disease is rather prevalent in this district and is observed almost weekly. It is quite possible that cases similar to the ones about to be presented, while rare, might lead a “frightened at the time” client to have the veterinarian in charge dispose of a valuable animal unnecessarily, and to his grief later; particularly if destroyed because the animal in question, not having bitten anything, need not be held for complete development of the disease and consequent brain sections for Negri bodies.

### CASE 1

*Subject:* Boston terrier, female; age, seven years; general condition, excellent.

*History:* According to the client, the subject was treated by a veterinarian for a mild otitis, with no observable change in the condition for one year. At the end of that time the dog was presented to the writer, who treated a right otitis media with apparently rapid response. The case was discharged and not heard from until one month later (September 12, 1932), when the owner noticed the animal presented a languid appearance and refused her night meal. The morning of September 13, the lower jaw was dropped, there was drooling from the lips and the animal did not appear to hear well. The dog was presented at the hospital that night.

*Symptoms:* Temperature, 103° F.; slight salivation, lower mandible relaxed, pupils partly dilated, severe right and slight

\*Received for publication, December 14, 1932.

left otitis media, ears partially drooped, hearing impaired, patient calm.

*Tentative diagnosis:* Suspicious of rabies.

*Treatment and observations:* The animal was confined in a roomy iron cage for observation. Food was consumed daily with difficulty. On September 16, or the fourth day of illness, without any observable change in the animal, we cautiously administered a sodium bicarbonate enema and unintentionally flushed her through. (This likewise occurred with ease on successive days.) Doses of 1/100 grain each of strychnin sulfate and atropin sulfate were given subcutaneously. Ineffectual attempts were made to stimulate reflex movements and teasing the mouth parts caused no fury or attempts to bite.

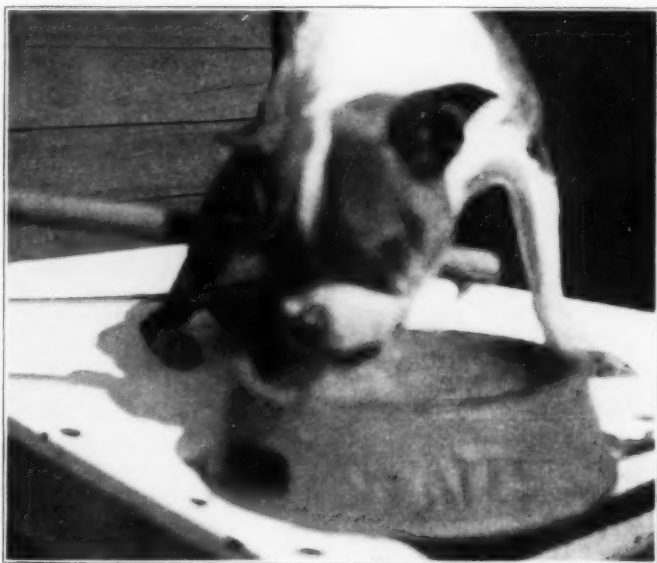


FIG. 1. Case 1, on thirteenth day of illness.

Thirst was apparent but upon attempting to drink water the subject would lap for minutes at a time, while the level of fluid remained the same, but always contained thick saliva on the surface. Meals were offered twice daily in the form of  $1\frac{1}{3}$  pound of cooked and raw beef respectively. There was slight difficulty in taking the food but the animal succeeded by pushing its muzzle into the pan, thus forcing the food to the posterior portion of the tongue. Appetite was always good. Temperature was never above 102.5° F. after the first day and often was normal.

On the eighth day we confined the animal and made a thorough examination of the mouth. A stomach-tube was passed freely and gastric lavage was negative. The mucous membranes of the pharynx and posterior surface of the tongue showed hyperemia and mottled discoloration typical of the rabid dog. The otitis media was treated at this time.

Authorities on rabies state that unless death ensues by ten days, in slow cases, we can eliminate rabies. However, in the November, 1932, issue of the *North American Veterinarian*, Pro-



FIG. 2. Case 1, recovered, 53 days after onset of illness.

fessor M. G. Lisi reports a case which died on the 17th day, whose brain was positive for Negri bodies. On the 15th day the present case indicated no change except less salivation. The animal was still very amiable. Rabies was eliminated. Specifically, details pointed to a partial paralysis of the laryngeal and pharyngeal branches of the tenth cranial or pneumogastric nerve; a complete paralysis of the inferior maxillary branch of the fifth cranial or trigeminus nerve and a complete paralysis of the eighth cranial or auditory nerve. (Discussion on experimental inoculation and recovery follows case 2.)

*Diagnosis:* Trigeminus, pneumogastric and auditory paralysis.

*Treatment:* Hypodermic doses of strychnin sulfate increased to 1/60 grain twice daily. Otitis media treated every four days

with 10 per cent silver nitrate and pellatol ointment, with marked amelioration.

#### CASE 2

*Subject:* Collie and Spitz, female; age, 5 years; condition, too obese.

*History:* Slight dropping of the lower jaw was noticed two days before presentation at the hospital. The dog appeared very



FIG. 3. Case 2, on fifth day of illness.

thirsty but had a failing appetite. The animal was brought to the hospital September 22, 1932.

*Symptoms:* Temperature, normal; lower mandible, relaxed; slight salivation, amorexia, polydipsia, intestinal toxemia. The animal was very amiable to speech and petting, showing no nervous tendencies.

*Tentative diagnosis:* Suspicious of rabies.

*Treatment and observations:* Sodium bicarbonate enema and

confined in an iron cage. On September 23 a meal of  $\frac{1}{2}$  pound cooked beef was consumed with difficulty. The animal lapped water frequently; the quantity remaining the same but the surface was covered with thick saliva. On the seventh day of illness, a saline cathartic was administered; also 1/100 grain strychnin sulfate subcutaneously twice daily. As temperature and evacuations were normal after this, no further treatment was attempted. Food was taken regularly. Salivation had decreased a bit. The animal exhibited no nervous tendencies. This being a mongrel dog, the owner did not care to have any expense incurred and left the dog at our disposal. This animal was subject to intestinal intoxication, which improved generally on correct diet and saline cathartics. On the tenth day of illness, marked improvement was noticed in the paralyzed mandible which assumed normal function by the twelfth day; also ability to drink appeared normal again.

*Diagnosis:* Trigeminal and pneumogastric paralysis.

#### DISCUSSION

Consultation was invited. Among other things it was suggested that infectious bulbar paralysis should be eliminated. The writer selected a six-year-old Airedale for inoculation. Two cc of fresh blood was drawn from case 1 and immediately inoculated subcutaneously over the left shoulder of the Airedale; also 1 cc into the left external saphenous vein. A similar inoculation was made with fresh blood from case 2 on the right side. That was October 3, 1932. Results were still negative two months later. The incubation period of infectious bulbar paralysis is two to nine days and intense irritation and later sloughing results at the point of inoculation. The causative agent is a filtrable virus present in the blood-stream.

On October 9, the mongrel (case 2) refused most of her meal. The nictitans membranes were slightly protruded, skin slightly toxic but the animal was alert. The intermittent intestinal intoxication was active again. The following day, no further change in the animal and no treatment having been offered, the dog was destroyed with saturated magnesium sulfate intravenously and autopsied to note the conditions at this stage.

*Postmortem report:* Productive gastro-enteritis, slight in the pyloric portion of the stomach, and severe in the entire small intestine, which was coated with a thick copious catarrh.

Slight interstitial nephritis.

Slight parenchymatous degeneration of spleen and liver.

The Boston terrier was discharged October 11, 1932, or 28 days after the onset of symptoms. The general condition was good, the otitis rapidly regressing, and the mandibular paralysis showed no improvement. The temporal muscles portrayed very marked atrophy of disuse.

Seven days later, the owner reported improvement in that the lower jaw was less relaxed. Seven days later, or 14 days after being discharged from the hospital, the animal was returned for the writer's observations. This was 42 days from the onset of illness. The sense of hearing showed marked improvement. The mandible had resumed normal function. The temporal muscles were quite sunken. A large bone in place of the regular meal was recommended twice weekly to stimulate strenuous mastication and exercise of those muscles. On November 4, 1932, the writer saw the animal and took a photograph. The atrophied temporal muscles were filling in rapidly. The previous otitis cleared up, the ears were more erect and the sense of hearing was quite acute.

#### CONCLUSIONS

Müller and Glass, likewise Brumley, refer to trigeminal paralysis in their texts as being the result of various etiological factors, among which are severe intestinal disturbances bringing about toxemias; inflammatory diseases of the ear and so forth.

In case 1 we had a single noticeable malady—otitis (probably secondary interna as well as media), which was rather severe and somewhat chronic in nature. Both trigeminal and pneumogastric nerves give off auricular branches in addition to the auditory nerve, which controls the sense of hearing. It seems possible that the inflammation and pus accumulation here may have caused sufficient intoxication temporarily to impair these and succeeding branches, particularly since response was parallel to the otitis amelioration.

In case 2 a chronic intestinal alteration of as severe a nature as found might cause sufficient putrefaction and intoxication temporarily to impair such nerve distribution.

#### ACKNOWLEDGMENTS

The writer wishes to express his sincere gratitude for the co-operation of the following consultants: Dr. W. J. Lentz, Professor of Anatomy and Director of Small Animal Clinics, University of Pennsylvania, Philadelphia, Pa.; Dr. Adolph Eichhorn, Director, Veterinary Department, Lederle Laboratories, Pearl River, N. Y.; Dr. W. E. Martindale, Rabies Investigation, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa.

## SIMULTANEOUS INFECTION OF COW AND FETUS WITH BLACKLEG\*

By J. E. SCHNEIDER and B. J. MCGROARTY

*Mulford Biological Laboratories, Sharp and Dohme  
Glenolden, Pennsylvania*

On April 18, 1932, a cow approximately 15 months old was injected intramuscularly on each rump with 5 cc of a pure blackleg culture and the cow died of blackleg in 32 hours.

On postmortem we found a fetus, probably 3½ to 4 months old, with a discolored blackish area the size of a pea on each rump without swelling or the presence of gas. The discolored area was dissected out and a piece of the tissue planted on liver-brain bouillon and incubated for 24 hours at 37.5° C. A good growth with much gas developed.

A small portion of the tissue was ground up in normal salt solution and 0.5 cc of the emulsion injected into each of three guinea pigs. All three pigs died in 24 hours with typical blackleg lesions. A heart-blood culture of each guinea pig was made on liver-brain bouillon and incubated for 24 hours. The cultures showed good growth and gas. Microscopic examination revealed bacilli typical of blackleg (*Clostridium chauvoei*).

## A CASE OF HYDRONEPHROSIS IN A DOG†

By PAUL C. UNDERWOOD, *Washington, D. C.*

*Zoölogical Division, Bureau of Animal Industry  
U. S. Department of Agriculture*

The animal in question was of Spaniel-Collie breeding, 3 years old, weighing 35 pounds, and to all external appearances was in excellent physical condition. The dog was being used in the course of some experimental studies and was noticed not because of any physical disability but because of its vigor and activity. The animal was particularly aggressive among its mates and showed no fear of physical contact or any tendency to favor an ailing part. It was apparent that if any pain or discomfort had previously accompanied the condition found at necropsy, such discomfort must have subsided completely by the time the animal was under observation.

The animal was killed at the conclusion of the experiment and a hydronephrosis of the right kidney was discovered at necropsy.

\*Received for publication, December 23, 1932.

†Received for publication, January 9, 1933.

A large cyst was found firmly attached to the dorsal wall of the abdomen. The cyst measured  $3\frac{1}{2}$  by  $5\frac{1}{2}$  inches, retained to some extent the contour of the kidney, and was smooth and tense from internal pressure. The blood-vascular system was limited to a network of vessels which were distributed through the kidney capsule, giving the cyst a somewhat lobulated appearance.

The increased pressure in the organ had entirely destroyed all the kidney tissue, and this tissue had been replaced by a large quantity of clear, amber-colored fluid. The ureter of the affected kidney was greatly enlarged, and measured one-half inch in diameter at its pelvic attachment. The diameter of the ureter was greatly lessened at the point of obstruction, which was about five inches from the renal pelvis. After taking a very tortuous

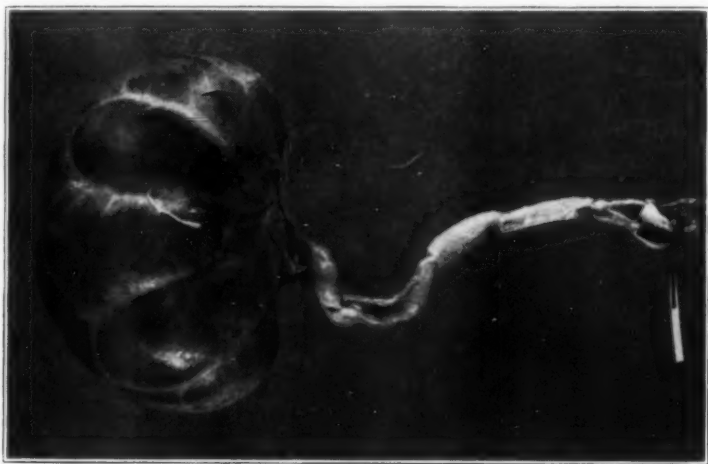


FIG. 1. Kidney of dog, showing hydronephrosis. Arrow indicates point of obstruction caused by calculus.

course for the first five inches, the enlarged portion of the ureter straightened and stopped abruptly at the constriction. Palpation of the ureter at this point revealed a calculus which had caused the obstruction. The calculus was of a caseocalcareous consistency, and was firmly wedged in the lumen of the ureter. The diameter of the ureter from the point of obstruction to the bladder was greatly reduced. (See figure 1.)

While the affected kidney gave no indication of functional activity, and the occlusion of the ureter was apparently complete, the left, or remaining, kidney gave no marked indication of response to its increased activity. Except for a very slight enlarge-

ment, the visible compensating hypertrophy which would be expected to occur in such cases was absent. Histological study of the kidney revealed slight hypertrophy. In proportion the ureter of the left kidney gave more indication of the activity of this organ than did the organ itself; the ureter was enlarged to a very marked degree. There was no obstruction or constriction of the left ureter to account in any other way for its increased diameter.

Hydronephrosis does not appear to be very common in dogs in the United States. It has not been observed at Washington, D. C., by Hall in the course of 23 years, or by Wright in four years. At Detroit, Michigan, Hall and Wigdor, as reported by Wigdor,<sup>1</sup> found it only once in 280 dogs examined over a period of two years.

#### REFERENCE

<sup>1</sup>Wigdor, M.: Pathological conditions found on necropsy of 280 dogs. *Jour. A. V. M. A.*, lxi (1926), n. s. 22 (3), pp. 357-361.

### AN OUTBREAK OF FOWL-TYPHOID IN GUINEA FOWLS (*NUMIDA MELEAGRIS*)\*

By E. P. JOHNSON and G. W. ANDERSON

*Department of Zoölogy and Animal Pathology  
Virginia Polytechnic Institute, Blacksburg, Va.*

Avian typhoid usually is considered a serious disease in domestic chickens and also to some extent in turkeys, but guinea fowls have been considered much less susceptible and, as far as we are able to ascertain, the literature does not describe outbreaks of this disease in guinea fowls.

Recently our attention was called to a disease occurring in a flock of about 100 of these birds, in which one or two had been dying daily without any symptoms noticeable to the owner. The death-rate seemed to increase and the owner supplied a bird for our examinations.

*Symptoms:* The only outstanding symptoms were paleness about the head and slight, yellowish-colored diarrhea. It had also been noticed that the birds would be somewhat droopy and lose their appetites a few days before death.

*Gross lesions:* The liver and spleen were congested and somewhat dark in color. There were also a few pin-point areas of necrosis on the liver. These organs, likewise, were enlarged and friable. The anterior portion of the intestines was hyperemic.

\*Received for publication, January 12, 1933.

with numerous minute hemorrhages noticeable in the somewhat thickened mucosa. The remaining organs appeared to have no gross lesions except a general appearance of congestion.

*Causative organism:* Smears from the liver and spleen, on both plain liver-infusion-agar plates and similar media containing brilliant-green dye, gave practically a pure culture of a small, non-motile, Gram-negative rod with the following carbohydrate fermentation reactions:

Dextrose—acid but no gas.	Sucrose—no acid or gas.
Lactose—no acid or gas.	Mannite—acid but no gas.
Dulcitol—acid but no gas.	Maltose—acid but no gas.

The fermentation reactions were checked in three series of tests.

*Biologic reactions of the organism:* Two rabbits were hyperimmunized by repeated injections of a known stock culture of *Salmonella gallinarum*. When the sera of these rabbits produced complete agglutination of the stock organism in a dilution of 1:6400, the rabbits were bled and their sera used to determine the agglutinating properties with the organism isolated from the guinea fowl, with the results indicated in table I.

TABLE I—Agglutination reactions with hyperimmunized rabbit sera.

DILUTIONS	1:200	1:400	1:800	1:1600	1:3200	1:6400	CONTROL
<i>Salmonella gallinarum</i>	+++	+++	+++	+++	+++	+++	—
Organism from guinea fowl	+++	+++	+++	+++	+++	+++	—

#### CONCLUSIONS

From the gross lesions observed and from the cultural and biological characteristics of the organism isolated, it may be concluded that this organism is identical with the causative agent of fowl-typhoid.

#### SUMMARY

This outbreak continued until about 30 per cent of the birds had died but was checked by prompt removal of the sick birds and by thorough cleaning and disinfection of the premises.

It is thought that this outbreak is of sufficient importance to be reported, as veterinarians and others might have difficulty in making diagnoses in similar outbreaks without resorting to careful laboratory examinations.



# REVIEWS

ANIMAL DISEASES IN SOUTH AFRICA.. M. W. Henning, Professor of Veterinary Science, University of Pretoria. 2 vol. xxi+878 pages, with 127 figures. Central News Agency, Ltd., Johannesburg, South Africa., 1932. Cloth, £2 10s.

This work of two volumes is a valuable addition to English veterinary literature. The books are the eleventh and twelfth volumes of the South African Agricultural Series, edited by H. D. Leppan, M. C., B. Sc. A., M. Sc.

In the foreword, Dr. P. J. du Toit points out that fifty years ago very little was known about animal diseases in South Africa. However, laboratory research workers and field observers have made valuable contributions to our knowledge of those diseases peculiar to South Africa. Among the laboratory workers Sir Arnold Theiler is the outstanding figure, and the work of Dr. Duncan Hutcheon is conspicuous among the clinicians.

The author has selected for treatment only those diseases that are of peculiar interest to South Africa. Volume I covers diseases caused by bacteria and protozoa. Volume II discusses diseases caused by filtrable viruses, those caused by poisonous plants, and deficiency diseases. More than half of the 127 illustrations in the two volumes are found in the part dealing with poisonous plants.

Thirteen bacterial diseases are covered in part I, a chapter being devoted to each one. A list of references follows each chapter. Naturally most of these are to articles by South African investigators, although many contributions by American workers are found in the bibliographies on Bang's disease, canine distemper, rabies and tuberculosis.

Part II discusses eight protozoal diseases. Of these, dourine, redwater (Texas fever) and gallsickness (anaplasmosis) are the only ones familiar to American veterinarians. Ten years ago, anaplasmosis would not have been included in such a list. This suggests that some knowledge of the other protozoal diseases would not be amiss. One or more of these diseases may make an appearance unexpectedly at any time.

Part III treats of fourteen virus diseases. Among these there are several that are all too familiar to the profession in this country, including hog cholera, canine distemper, foot-and-mouth disease and rabies. One other, contagious pleuropneumonia, was known to earlier generations. Equine infectious anemia is not included among the filtrable virus diseases of South Africa.

Part IV contains 21 chapters, each dealing with a family of plants causing trouble to live stock. Descriptions of the plants are given, with symptoms of poisoning and methods of treatment and prevention, if known. This part is profusely illustrated with excellent drawings and photographs of plants and affected animals.

Part V consists of a chapter on domsiekte, a deficiency disease. Lamsiekte and styfsiekte, also deficiency diseases, are discussed in other parts of the book, due to their complex etiologies.

The author is to be congratulated on the clear, concise and systematic manner in which he has presented his subject and the publishers are to be complimented on the mechanical features of the work.

### PUBLICATIONS RECEIVED

- Bacterium Abortus* Infection in the Fowl II. Herbert L. Gilman and E. L. Brunett. Reprint from Rpt. N. Y. State Vet. Coll. for 1930-1931, pp. 149-166.
- Enzoötic Haematuria (Haematuria Vesicalis) of Cattle in South Australia. L. B. Bull, C. G. Dickinson, and A. T. Dann. (Pamph. 33, Coun. for Sci. & Ind. Res., Commonwealth of Australia. Melbourne, Australia, 1932. pp. 24.)
- Studies on Bovine Mastitis. VII. The Serological Characters of Mastitis Streptococci. A. W. Stableforth. Reprint from *Jour. Comp. Path. & Therap.*, xlv (1932), pt. 3, pp. 185-211.
- Parasites of Minks and Their Control. Karl B. Hanson. (Bi-1235, Bur. Biol. Survey, U. S. Dept. Agr. Washington, D. C., 1932. pp. 7.)
- U. S. Army, Ann. Rpt. of Surgeon General for Year Ending June 30, 1932. (U. S. War Dept. Washington, D. C., 1932. pp. 336.)
- Studies on the Nutritive Value of Kelp Meal for Animal Feeding. H. P. Morris. (Tech. Rpt. 5, Bur. Fisheries, U. S. Dept. Comm. Washington, D. C., 1932. pp. 23.)
- Some Tick Investigations in Kenya Colony. E. Aneurin Lewis. Reprint from *Parasitol.*, xxiv (1932), 2, pp. 175-182.
- Oesophagostomum Multifoliatum* N. Sp. An Undescribed Nematode from Sheep and Goats. R. Daubney and J. R. Hudson. Reprint from *Parasitol.*, xxiv (1932), 2, pp. 265-267.
- Southern Rhodesia, Abridged Report of Director of Veterinary Research for 1931. E. W. Bevan. (Salisbury, Rhodesia, 1932, pp. 3.)
- Studies in Louping-ill (An Encephalomyelitis of Sheep). II. Transmission by the Sheep Tick, *Ixodes Ricinus* L. J. MacLeod and W. S. Gordon. Reprint from *Jour. Comp. Path. & Therap.*, xlv (1932), pt. 3, pp. 240-256.



# ABSTRACTS

SIXIEME ASSEMBLÉE DES VÉTÉRINAIRES ALLEMANDS, POUR LA LUTTE CONTRE DES MALADIES DE L'ELEVAGE, TENUE A LEIPZIG DU 3 AU 6 OCTOBRE, 1931 (Sixth Conference of German Veterinarians, for the Campaign Against Diseases of Reproduction, Held at Leipzig, October 3-6, 1931). Abst., Rev. Gén. de Méd. Vét., xli (1932), 487, pp. 434-435.

*Report of Professor Miessner:* Infectious abortion in mares has almost completely disappeared from Germany. Out of 43 cadavers of colts examined, 70 per cent were dead from pyosepticemia. Much of the sterility can be charged up to defective alimentation. Researches concerning Bang's disease of cows were carried out on 80,678 cases. Some 7,000 calves were examined for diseases of the newborn, with the result that bacillary diarrhea and faulty feeding were found to play the principal rôle.

*Report of Professor Ziegler:* Systematic researches on sterility were carried out annually on 26,000 bovines. In Saxony, 20 to 30 per cent of the herds are affected with Bang's disease. The abortions are lowered from 20 to 6 per cent, where one to two vaccinations with living cultures is practiced. The best results are obtained when the vaccinations are done 6 to 8 weeks after service. The vaccination of cows, soon after they have aborted, gives but little benefit. *Sterility is not much more prevalent in infected than in non-infected herds!*

*Report of Professor Zwick:* At the laboratory, as in the field, vaccination 6 to 8 weeks after service gives a durable immunity. The official figures are as follows: From killed-culture vaccine (bacterin) abortions were decreased from 25.3 to 15.6 per cent and from live-culture vaccine from 28 to 6.36 per cent. Preference should be given, therefore, to the live-culture vaccine. Although Little and Smith have obtained good results from a single vaccination, it should be repeated at different periods of gestation. Systematic vaccination in Pomerania and Lower Silesia decreased abortions from 22 to 6.6 per cent. The benefits derived from hygienic measures should not be ignored.

In Germany, 600 cases of Bang's infection were observed in

man. The contagion occurs chiefly from direct contact. In the rural districts more cases were observed in men, while in cities the percentages of men and women were about equal. Physicians who claim that these infections are due to live-culture vaccinations should prove it. Schuman has shown that as many cases can arise from non-vaccinated herds as from the ones that were vaccinated.

In herds only slightly infected, Bang's disease can be eliminated by hygienic measures.

*Report of Professor Götze:* The purchase of cows must be supervised and breeders must be educated to this advice. Aborters should not be bred again for three months. Heifers may be bred twelve weeks and cows eight weeks after vaccination. All nostrums sold for the cure of Bang's disease should be suppressed.

*Report of Professor Jensen:* In the Scandinavian countries researches have been carried out on 200,000 cases of Bang's disease, but opinions still differ on the question of appropriate measures to apply, except that all are in accord on the value of hygienic practices.

*Report of Dr. Benixin:* Working with Professor Bang, a study was made on the skin as a portal of entrance for *Br. abortus*. It was found that the intact skin is permeable to that organism.

L. A. M.

LA VACCINATION ANTITETANIQUE EN MÉDECINE VÉTÉRINAIRE (Antitetanic Vaccination in Veterinary Medicine). J. Verge. *Rev. Gén. de Méd. Vét.*, xli (1932), 487, pp. 407-411.

Artificially induced active immunity against tetanus in domesticated animals has been a demonstrable fact since the work of Ramon and Descombey, which led to the production of tetanus anatoxin, now made by simply adding 2.0 cc of formalin to a litre of the specific toxin and incubating the mixture at 40° C. for a month. The finished product consists of 10 cc of the attenuated liquid mixed with tapioca powder. At the Pasteur Institute the finished mixture is placed in 10-cc ampules for future use.

Two injections are required to assure a lasting or lifetime immunity. The injections are made one month apart on the side of the neck. At the Pasteur Institute at Garches (near Paris), among a thousand horses thus immunized since 1925, no cases of tetanus have developed. Among 557 horses thus vaccinated in the French Army since 1927, there were no cases of tetanus, while among 953 non-vaccinated horses included in the

observation there were four mortal cases. In 1928, in one regiment, 218 vaccinated animals showed no cases of tetanus, while among the 473 non-vaccinated, two non-fatal cases developed.

But the crowning proof of efficacy was the observation of 1929, when approximately 35,000 horses thus were immunized. In this large group the average morbidity from tetanus before vaccination was 4.55 per cent, with a mortality of 3.98 per cent of the total number. After the animals received the immunizing injections, only three mild cases of tetanus developed, that is to say, the mortality dropped to zero and the morbidity to 0.6 per cent.

The author quotes various practitioners who assert that they have seen no cases of tetanus develop in their vaccinated animals.

Cattle receive the same dose as horses, and dogs one-half as much. In valuable horses and in places where tetanus is unusually prevalent, an annual vaccination of one 10-cc dose may be employed as an additional precaution.

L. A. M.

LES NAUFRAGEURS DU PROGRÈS (The Wreckers of Progress).

Clément Roéland. *Rev. de Path. Comp.*, xxxii (1932), 429, pp. 675-679.

The author, who is both physician and veterinarian in education and member of the city council of Paris, has been relentless in his campaign for a better control of the milk supply of that city and pleads again for public support of measures leading to an improvement in the quality of raw milk as compared with attempts to render dirty milk safer by pasteurization. The author reviews the hostility his recommendations have brought from the agricultural side of the debaters whose followers he criticizes as "wreckers of progress," to their own disadvantage and to whom he dedicates a verse:

There are pathogens in milk.  
Pasteurize and drink!  
There is pus in milk.  
Pasteurize and drink!  
There is x in milk.  
Pasteurize and drink!

This the author characterizes as the "cream of the enemy's effort" to purify the milk supply of the French people, and hopes that in the near future their American hosts, lovers of raw milk, will not be compelled to bring clean milk from England, as some now insist upon doing, to avoid drinking the questionable milk supply of the French metropolis. He points out that while there

are intelligent, courageous and conscientious producers who realize the importance of reforms and practice them, it is well known that others more numerous paralyze good intentions and sap energy to protect the existing routine. The author pleads for public support of the efforts being made in this connection and points to certain districts of France, Holland, Belgium, England and the United States where raw milk is rendered safe through the instrumentality of properly enforced hygienic measures among the producers, as was stated at the International Milk Congress held in Paris in 1926.

L. A. M.

VACCINATION WITH BCG OUTSIDE OF FRANCE. *Jour. Amer. Med. Asso.*, xcix (1932), 15, p. 1275.

Authorities outside of France (England, United States, Canada, Brazil, Uruguay, Roumania, Yugoslavia, Poland, et al) are quoted to the effect that excellent results are being obtained in protecting both man and animals against tuberculous infection through the instrumentality of BCG vaccinations. "BCG vaccination is certainly harmless and confers on the organism resistance to tuberculous contagion," is the conclusion drawn from the various papers written by the authors cited. The author emphasizes the work of Park (U. S.), of Gerlach (Austria), and of Assis (Brazil), who admit of no doubt that unfavorable conclusions have been based upon "error of observation and faulty technic," and he furthermore asserts that the tuberculosis vaccine of Calmette and Guérin protects the vaccinated subject against contamination and virulent superinfections beyond any question of a doubt. Statistical reports indicate that general child mortality has diminished where BCG vaccination has been practiced on a large scale, notably in Sweden, Poland, Roumania and Uruguay.

L. A. M.

TRAITEMENT D'UN CAS D'HEMOPHILIE CHEZ UN CHIEN (The Treatment of a Case of Hemophilia in a Dog). *Noell. Rec. de Méd. Vét.*, cviii (1932), 6, p. 356.

The patient was a German shepherd affected with penial polypi which the author extirpated in the usual manner but under profound anesthesia, owing to the intractable nature of the subject. Three polypi were removed but from the moment of ablation the bleeding was stubborn, failing to yield to ordinary styptic applications. The dripping continued, drop by drop, all night. Blood

collected from the droppings failed to show any tendency to coagulate even when mixed with a 5 to 10 per cent solution of antipyrine, nor were any helpful results obtained from injections of a cubic centimeter of fluid extract of ergot. Neither were any benefits derived from injections of artificial serum and sodium cacodylate.

As there was no normal serum available, the author thoughtfully substituted tetanus antitoxin. Two injections of 10 cc were made. The hemorrhage ceased promptly and did not recur.

L. A. M.

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L'AVORTEMENT EPIZOOTIQUE A BRUCELLA CHEZ LES PORCS AU DENEMARK (The Brucella Type of Epizootic Abortion in Swine in Denmark). A. Thomsen. *Rev. Gén. de Méd. Vét.*, xl (1931), 476, p. 457.

The author had occasion to study an epizootic of contagious abortion of sows in 1929. He was able to demonstrate how the disease was propagated in Denmark.

The causal organism was isolated in the sexual organs of six boars out of ten examined.

The percentage of abortions among infected sows was lower than is usual for cows but, nevertheless, the economic importance of the disease was great.

The author was not able to demonstrate the transmission of the infection to cows or vice versa. By making 5,600 serological tests of sows and boars in abattoirs to determine the incidence of the disease in Denmark, the negative findings showed that the swine type has more the characteristic of a transient outbreak as compared with its fixity in the bovine species.

L. A. M.

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### New State Veterinarian for Arizona

Governor B. B. Moeur, of Arizona, appointed Dr. R. J. Hight (Chi. '07), of Tempe, as state veterinarian to succeed Dr. Edward L. Stam (Wash. '20), whose term of office expired December 31, 1932. Dr. Hight served as state veterinarian until January 21, but resigned on that day as a result of the failure of the State Senate to confirm his appointment. Dr. C. T. Guilfoyle (U. P. '11), of Douglas, then was given the appointment, according to word received from Dr. J. C. McGrath, A. V. M. A. resident secretary for Arizona, but the latter did not indicate whether Dr. Guilfoyle's appointment had been confirmed or not.



### Regular Army

Orders assigning Major Louis G. Weisman to duty at Fort Benning, Ga., upon completion of his present tour of foreign service, are amended to assign him to duty at Fort Clark, Tex.

Orders assigning Captain Laurence R. Bower to Fort Bliss, Tex., upon completion of his present tour of foreign service in the Philippine Department, are amended so as to assign him to duty at the New York port of embarkation, Brooklyn, N. Y.

Major Joseph H. Dornblaser, upon relief from assignment and duty at Fort D. A. Russell, Tex., by the commanding general Eighth Corps Area, is assigned to Fort Bliss, Tex., for duty.

First Lt. Harvie R. Ellis, upon relief from assignment and duty at Fort D. A. Russell, Tex., by the commanding general Eighth Corps Area, is assigned to Fort Riley, Kan., for duty.

### Veterinary Reserve Corps

#### *New Acceptances*

Chew, Roy D., Capt., Covington, Ind.

Case, Ralph Wilson, 2nd Lt., 722 Miller St., Prescott, Ariz.

Neal, Charles William, 2nd Lt., 1619 S. Laredo St., San Antonio, Tex.

Orson, Oliver Wilton, 2nd Lt., 410 W. Illinois (Box 455), Midland, Tex.

Pease, Fred W., 2nd Lt., 910 E. 6th St., Austin, Tex.

Rackley, Ernest William, 2nd Lt., 115 Stone St., Waynesboro, Ga.

#### *Promotions*

##### **To**

Westerberg, Ralph Victor, Capt., 403 W. Main St., New Britain, Conn.

Miller, William Colter, Major Aux-Res., 88 Minnesota Ave., Long Beach, N. Y.

### Seize Worm-Infested Fish

Six carloads, aggregating 95,000 pounds of wormy, frozen "mongrel whitefish" (tullibeas), were seized recently by federal officials at Baltimore, Md. The fish were shipped from the Lake of the Woods, Minn., region to cold storage warehouses in Baltimore, and were to be smoked and sold to consumers in the Central Atlantic States. Because of the presence of parasitic worms encysted in the flesh of the tullibeas, the fish were classified as adulterated under the national Pure Food and Drugs Act and subject to seizure.



## MARYLAND STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Maryland State Veterinary Medical Association was held at Medical Hall, Baltimore, December 8, 1932, with 55 members present. Dr. W. E. Campbell's presidential address carried so much food for thought that it is being reported in detail. It was substantially as follows:

In appearing before the Maryland State Veterinary Medical Association, the question arises, "Why we are here and what is the object of *this* gathering and the purpose of this Association and other similar organizations?" We are not basically a social organization nor a fraternal order, nor yet a political organization, though we gain some experience in the work of these lines of endeavor. No, the reason for the existence of an organization of this kind is for advancement and progress; advancement of the individual socially, mentally, and I may say morally, and the advancement of the profession. When such an organization is properly functioning, only progress can obtain.

We are searching for the truth and when false statements are presented to us, they must and will be turned down, passed over and be forgotten, possibly not at once, but as soon as they are recognized. We cannot thrive on anything else. This is possibly one of the reasons we are so helpless in the political arena where subterfuge seems to be in vogue.

The past year has not been particularly kind to us. But taking our profession as a whole and in all its branches I believe we have resisted the hardships and deprivations of the depression as well as any other profession and much better than the average class and wage-earner. Many times in the past two years I have been more than thankful that I had something in my head that couldn't be lost on the stock market nor be levied on by the sheriff.

The present times are the best advertisements for the schools and colleges. They offer a young man or woman something of which they cannot be robbed. This has filled our veterinary schools and assures us greater progress in our profession than most of us can visualize. This is happening in spite of the deplorable condition of the farmer, dairyman and live stock industries upon whose prosperity our life-blood depends.

The practitioner has been hurt and some of us badly, but as yet I know of but few who have entered the bread-line or suffered for the necessities of life. Some still enjoy luxuries well above the average. I am not ordinarily optimistic, but, on looking back over the last fifty years and reviewing the forward strides our profession has made from the zero mark to its present state, I cannot

help but view with delight the prospects of our future. Such examples are numerous in other nations where the profession is older, even though their economic conditions are and always have been worse. In practically all European countries the veterinarian is held in high esteem and only last month I read an article in the *National Geographic Magazine* which stated that in little known Bulgaria the veterinarians had a higher social standing and a better community rating than even the medical profession.

It is not altogether our rapid advancement that makes the future so bright but also the veterinarian's adaptability. In one generation we have shifted from almost one hundred per cent equine specialists to diversified fields not even imagined twenty years ago. Cows, dogs, cats, pigs and sheep far outnumber the horses in most sections of the country. But even the horse is coming back. Prevailing prices have made the farmer think twice before he sells his corn for 20 cents or less a bushel and pays 20 cents a gallon for gasoline when he can get a dollar's worth of fuel from the corn by burning it in a good horse or mule.

But there are other things that stand in our way besides the general economic conditions. We have enemies outside our profession both intentional and unintentional. Some localities are more harassed than others and the breeds differ. There have been complaints again of county agents whose minds seem to run toward the bistoury and syringe rather than organization and insects. In this particular species, I am always suspicious that some of their insects have escaped the spray-gun and have entered their heads with the resulting miscarriage of their sense of duty. I have always been glad that they chose their profession instead of ours. What few county agents I have known can be pretty well classed according to their regard toward the veterinary profession. Those who have advanced have all been friends of the veterinarian and have made work for them. Those who have just stayed county agents don't attempt veterinary work and neither help nor hinder it. They are probably lazy. And those who have tried to practice our profession as part of their work have eventually disappeared as county agents. So the county agent has ceased to worry me long at a time and I hope the rest of you will be as fortunate.

We also seem to have some political enemies which may or may not disappear with the return of freer money and less agitation on the tax problems. But this class can do our profession untold harm and undo at one stroke work accomplished only by years of study and unremitting perseverance. This class has to have some one to jump on in times like these, if for no other reason than to make a noise, and they naturally pick the weakest opponent. So when the smoke clears away the rebound will not hurt so badly. It behooves us to watch every move made by the executive and legislative bodies from the federal government down to the smallest municipalities. It is unfortunate that we are not financially able at this time to have a man on full time to keep his ear to the ground and warn us of any impending disaster. But as it is, the veterinarian will have to be his own watchdog and the society, I am sure, will be glad to do what it can to protect the rights of any branch of our profession.

This naturally leads to the matter of dues. It is probably a poor time to mention this subject but it is a condition that must be faced if we are to get anywhere. Our dues are ridiculously low. So low that it is and will be impossible to carry a balance of any

size to meet emergencies. It is not a mercenary or vague idea that organizations must have money to prosper and for protection. There is probably not a member here who does not belong to some fraternal order or maybe several, the dues of which many times exceed those paid into the treasury of this Association, and if we compare the benefits derived from the dues I am sure the balance will be in favor of the Association and would still be so if our dues were doubled or quadrupled. Secondly, while on this subject, I wish to call attention to the fact that the Association derives no benefit from them unless they are paid. So I plead with each and every member to pay his duties promptly and if you are in arrears to pay up, and I can assure you that you can never make a better investment. I only wish our treasury was full to overflowing and our dues large enough to start a sinking fund. In the future, such a fund might well be the difference between winning and losing battles we have to fight for our very existence.

It is a satire that the manufacturers of dog foods can spend millions in advertising their wares in the newspapers, periodicals and over the radio. The profession, to whom these same dogs largely owe their very existence, cannot afford more than a mere fraction of this amount of publicity to bring the facts before the people who are ignorant, to a large extent, of our very being. Such knowledge should state what we have done, are doing and can do for their pets and live stock if we are given an opportunity.

The first step toward a Utopia is in securing an active membership of one hundred per cent of all eligible and desirable veterinarians in the territory over which the organization has jurisdiction. It is only right and proper that this should be, as the members of the Association in no way derive more benefits from the results of our labors than do the non-members. They are even invited and do, to some extent, attend our meetings and profit thereby. The psychology that will let them accept continued favors and still get writer's cramp when it comes to signing on the dotted line for membership and for dues, is a mental state I cannot understand.

To study this condition and remedy it as far as possible, I suggest that the Association approve a committee, to be called the Membership Committee, to head this work. Further, let every member constitute himself a lone committeeman to learn why veterinarians of his acquaintance who are not members do not join the Association and report the fact to the committee, so that any differences may be smoothed out if possible and new members be obtained.

And one final suggestion: I have noticed that during the meetings the individuals most active in the discussion of problems that come up are members who have had some active office in the Association, especially ex-presidents and Executive Committee members. There is a reason for this that only those who have held one of those positions can appreciate. It is that they understand the functioning of the organization better and recognize the importance of matters being discussed more clearly and quickly than do those who have not had the opportunity to help govern. As free discussion on the floor by the members is the best tonic we can take, I believe it would be advisable to increase the number of members who have had the experience of the executive meetings by a more rapid rotation of officers. This can be accomplished by the elimination of the two-year term for president, which seems

to have become established by precedent, and including the vice-presidents on the Executive Committee. Further, the Executive Committee meetings might be opened to any member who desires to attend.

\* \* \*

It would not be fitting to close without a word about our Ladies Auxilliary, which has already been a great help to us, both as individuals and as an organization. I hope to see it grow until it outnumbers its parent Association. So I ask our members to do their part by bringing the ladies to the meetings and giving them at least an opportunity to enjoy the delightful society of the Auxilliary. I am doing my part as I have two prospective members coming along. I hope you all will do as well.

The second paper was entitled, "A Study of the *Brucella Abortus* Agglutination Titres of the Various Species of Farm Animals in Maryland," and was presented by Dr. L. J. Poelma, College Park.

The third paper was entitled, "The Tuberculin Testing of Accredited Herds in New York State," prepared by Dr. Charles Linch, Albany, N. Y., and read by Dr. R. C. Reed in the absence of the author. The high points of the paper are quoted as follows:

While the retesting of accredited herds was carried on at the owner's expense, it was often difficult to have the herd retest made when due. The charge for retesting accredited herds was not uniform; some veterinarians charged fifty cents a head, or less, while others charged one dollar a head, and some often considerably more.

After considerable correspondence with the owner relative to a retest of his accredited herd by an accredited veterinarian, very often it was necessary either to designate an official veterinarian to go out to his territory to make the retest, or wait until area testing was taken up in his locality.

In some of the counties it was found that not more than 10 per cent of the accredited herds were being tested under this plan, while in other counties the percentage being tested was considerably higher, perhaps 50 per cent or more.

In March, 1930, the Legislature made available a fund of \$400,000 for the retesting of fully accredited herds, at state expense. The work was started March 17. A similar appropriation was made during the 1931-1932 session of the Legislature. These tests are made by accredited veterinarians under the zone plan, each resident accredited veterinarian being assigned to a zone made up of one or more townships, the veterinarians working under a blanket authorization issued by the Commissioner of Agriculture and Markets.

The veterinarians in a county call a meeting at some central point, and agree on a satisfactory division of the territory. At the present time, 44 counties in the State have been divided into a number of zones or areas, each zone being made up of one or more townships, an accredited veterinarian being assigned to each zone. The veterinarian is supplied with a blanket authorization to retest all fully accredited herds in his zone, at state expense.

The rate of compensation agreed upon was \$2.50 for each herd tested, plus thirty cents for each cow in the herd. This would be \$2.80 for a herd of one animal, and \$5.50 for a herd of ten animals. To determine the amount of compensation for a herd, multiply by thirty cents the number of animals in the herd and add \$2.50, the herd price.

This sliding scale of compensation was worked out with the idea in mind of taking care of the small herd, as well as the large one. By allowing a herd charge, there is an inducement for the accredited veterinarian to retest the small herds which are often scattered through regions which have poor roads, as well as the large herds located on state roads.

On August 1, 1932, the price per animal was changed from thirty cents to twenty cents, the herd price remaining the same, \$2.50. This would be \$2.70 for a herd of one animal, and \$4.50 for a herd of ten animals.

Following this paper a general discussion apparently crystallized into the thought that the present method of tuberculin-testing of accredited herds by the accredited veterinarians in Maryland is producing satisfactory results. If, however, the farmers' organizations decide to introduce a bill in the coming Legislature, requiring this work to be done at state expense, it was felt that a modification of the New York system should be adopted.

The fourth paper consisted of case reports entitled, "Three Cases Illustrating the Value of Postmortem Examinations," presented by Dr. J. P. Turner, Washington, D. C. A case of necrobacillosis and two neoplasms in cattle were described. The symptoms were more or less misleading in each case and in the absence of postmortem examinations serious mistakes would have been made. The last paper presented was entitled, "Intravenous Therapy," by Dr. E. B. Dibbell, Baltimore.

During the business meeting, the advisability of introducing a veterinary practice bill at the coming meeting of the Legislature was discussed, as was also a state meat inspection bill. The Association approved the proposed plan of affiliation of state, territorial and provincial associations with the American Veterinary Medical Association.

The following officers were elected for the coming year: President, Dr. W. E. Campbell, Bel Air; first vice-president, Dr. R. C. Reed, College Park; second vice-president, Dr. F. S. Wharton, Centerville; secretary-treasurer, Dr. E. M. Pickens, College Park (reëlected); member of the Executive Committee, Dr. T. A. Ladson, Olney.

E. M. PICKENS, *Secretary.*

## NEBRASKA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-fifth annual meeting of the Nebraska State Veterinary Medical Association was held at the Hotel Yancey, Grand Island, December 13 and 14, 1932, with approximately 100 members and guests attending.

The meeting was called to order by Dr. C. C. Hall, President. The minutes of the previous meeting were approved as read. The President's address summarized activities during the year and included recommendations for the future. Dr. Hall has been untiring in his services and the Association is fortunate in having him as Chairman of the Executive Board for 1933, which is a legislative year.

Dr. W. T. Spencer, Chairman, indicated that the Legislative Committee would be active during 1933 in protecting the interests of the veterinary profession, in so far as lies within their power, and in recommending additional legislation favorable to the profession.

The Publicity Committee, Dr. A. H. Francis, Chairman, recounted their activities of the year, which included letter contest for 4-H Club members on "Why Farmers Should Raise Their Own Horses"; Veterinary Conference, March 8-10, Lincoln; and 20 sectional meetings over the state during the spring of 1932, every practitioner being invited to participate in abortion control work.

The Committee on Resolutions, Dr. L. V. Skidmore, Chairman, presented seven resolutions, all of which were adopted. Particular reference is made to resolution 4, as follows:

WHEREAS, After due consideration by the Executive Committee of the Nebraska State Veterinary Medical Association relating to attendance at sessions of this Association, therefore, be it

*Resolved*, That beginning with the 1933 meeting of the Nebraska State Veterinary Medical Association all sessions will be limited to paid-up members of this Association, and be it further

*Resolved*, That those residing outside of Nebraska and desiring admittance to sessions of this Association may do so by presenting visiting cards to the secretary of this Association, and be it further

*Resolved*, That this shall not apply to those who are invited by the proper officers of this Association to appear on our programs.

It was felt that this resolution was necessary to stimulate a more personal interest in the Association and to curtail a growing tendency of lending only moral support, leaving to a few the financial burden of carrying on the work. Attendance at annual meetings is desired and urged, but those enjoying Association

privileges should be active and contributing members, except as otherwise specified.

The first paper on the literary program, entitled "Anthrax," was presented by Dr. L. I. Hines, of Spencer. Dr. Hines practices in the anthrax territory and has had much practical experience in handling this disease. The subject was timely and well presented.

Mr. L. I. Frisbie, Lincoln, State Extension Agent, Boys and Girls Clubs, discussed, under the title "Another Live Stock Booster," coöperative measures which could well be adapted to the advantage of the veterinarian in giving more attention to 4-H Club work.

A telegram was received from Dr. C. H. Covault, Iowa State College, Ames, Iowa, who was on the program for the afternoon session for a discussion of "Cattle Practice," stating that he was unable to be present because of illness. Dr. J. D. Ray, of Kansas City, Mo., kindly substituted with an extemporaneous talk on laboratory technic.

Dr. Earle G. Johnson, Grand Island physician, addressed the Association on "Preventable Diseases Common to Man and Animals." He urged full coöperation in this direction between veterinarians and physicians, stressing prevention rather than cure in the following words:

Of course we can't prevent all diseases, but we can prevent some of them. The prevention of any disease carries with it more honor to the man who accomplished it than it would if the same man had cured thousands after they had contracted the disease.

In discussing Dr. Johnson's address, Dr. R. R. Dykstra, of Kansas State College, Manhattan, Kan., presented the viewpoint of the veterinarian in a very able manner and enumerated other communicable diseases, with scientific information regarding them, not mentioned by Dr. Johnson.

An illustrated lecture by Dr. Dykstra on "Recent Developments in Animal Surgery" was highly instructive and handled in the usual interesting and able manner characteristic of this speaker. The Association is deeply indebted to Dr. Dykstra and his associates for the coöperation they have always given in contributing to our programs.

Dr. J. P. Scott, of Kansas State College, was the next speaker on the program, his topic being "Results of Some Recent Investigations of Hemorrhagic Septicemia." Dr. Scott has done considerable research in this field and handled the subject in a scientific manner.

The evening was devoted to the annual banquet which was presided over by Dr. Frank Breed, of Lincoln, as toastmaster, who introduced the distinguished visitors. Dr. Dykstra, immediate past president of the American Veterinary Medical Association, gave the address of the evening. The balance of the evening was spent dancing and visiting among friends.

At the Wednesday morning session Dr. H. L. Feistner, Chief, Nebraska Bureau of Animal Industry, Lincoln, gave a report of the activities of his office, mentioning the fact that 38 per cent of all tuberculin testing done in 1931 was by resident practitioners. He offered recommendations urging organization of smaller groups of veterinarians, with educational meetings to be held once each month.

Dr. C. R. Collins, practitioner of Osceola, presented an interesting paper on "Poultry Practice." The preamble graphically outlines the veterinarians' position in poultry husbandry as follows:

Poultry practice is a phase of veterinary medicine that is receiving increased attention and it has been and will be one of the most profitable branches of the live stock industry.

Poultry husbandry is an extremely large field in itself and when thoroughly established affords great opportunities for the practitioner.

The success of such an industry depends largely upon disease control which must come through the veterinary profession.

Due to rapid development with great concentration in number of birds, over-production, lowered vitality, sooner or later result in disease infection, parasitism and malnutrition. Consequently the losses from the above-mentioned conditions are enormous.

The numerous problems of the poultryman demand a valuable service of the veterinary profession, more especially when confronted with increased prevalence of disease.

The subject "Small Animal Practice," by Dr. E. J. Frick, Kansas State College, was presented in an interesting and informative manner, as indicated by the lively discussion which followed.

"Practical Methods of Milk Inspection," by Dr. G. H. Mydland, practitioner of Horton, Kan., was one of the most valuable contributions to the program. Dr. Mydland has been highly successful in this line of work as applied to communities of 3,000 to 4,000 inhabitants and it is a phase of work which has been neglected in most localities and in which the veterinarian should take a conspicuous lead.

At the Wednesday afternoon session Dr. Frank Breed lectured on "Swine Erysipelas." His remarks included the following significant statement:

Bear in mind that the outstanding disease in swine in the Corn Belt is hog cholera. The safest way is to protect herds from cholera by vaccination and in suspected cases of swine erysipelas, send in the spleen, kidney, heart and liver to the laboratory . . . The final diagnosis on swine erysipelas must be in the laboratory. It cannot be done in the field at the present time.

During the past year, 141 sets of cultures for swine erysipelas have been sent in from Nebraska and of these, 63 have proven positive.

When Dr. J. S. Anderson, of Hastings, rose to tell of his "Forty Years of Service," the audience rose to their feet in splendid tribute to the high esteem in which he is held. For an hour he entertained the Association with a vivid description of his experiences, beginning 40 years ago when he entered practice, down to the present time. Dr. J. D. Sprague, of David City, one of his old colleagues, followed with appropriate remarks, concluding one of the best literary programs enjoyed for a number of years. These two men were honored by the Association in 1929 by being elected to honorary life membership.

The following officers were elected for the coming year: President, Dr. J. E. Weinman, Lincoln; vice-president, Dr. S. W. Phillips, David City; secretary-treasurer, Dr. E. C. Jones, Grand Island (reëlected). Drs. F. H. Oberg, Osceola, and Frank Breed, Lincoln, were elected to the Executive Board, to fill vacancies caused by the retirement of Drs. D. E. Trump and L. I. Hines.

Lincoln was the unanimous choice for the 1933 convention.

E. C. JONES, *Secretary*.

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### UNIVERSITY OF PENNSYLVANIA VETERINARY CONFERENCE

The annual Conference of Veterinarians which was held at the University of Pennsylvania, January 4 and 5, 1933, was well attended by veterinary practitioners and men in other branches of the profession coming from near and far. The program began with a session commencing Wednesday, January 4, at 1:00 p. m., with Dean G. A. Dick presiding. Dr. M. A. Emmerson, of the Department of Surgery, demonstrated a practical method of performing a rumenotomy under local anesthesia, with the animal standing, and this was followed by a practical demonstration of the most advanced technic in cauterization under local anesthesia, by Dr. W. J. Lee, also of the Department of Surgery. One man present expressed the feeling he had been amply repaid for making the trip to Philadelphia after witnessing these practical operations.

Following the surgical demonstrations there was a paper delivered by Dr. John G. Hardenbergh, of Plainsboro, N. J., on the subject of "Bang Disease Control in Large Herds," which was received with much interest, several men having questions to ask Dr. Hardenbergh and many commenting on the valuable report. Dr. Joseph McFarland, professor of pathology in the School of Medicine, then delivered a most entertaining lecture concerning "Death, Somatic and Molecular," thereby captivating the interest of everyone present by his eloquent and forceful delivery. This lecture was succeeded by an equally interesting and most informative paper by Dr. C. C. Palmer, of the University of Delaware, on the subject of "Colon Bacillosis and Hemorrhagic Septicemia of Calves," which terminated the afternoon session.

The evening session, with Dr. R. S. Amadon in charge, commenced with a lecture by Dr. Herbert Fox, professor of comparative pathology, on forms of tuberculosis in the lower animals, which was followed by a most interesting discussion by Dr. Arthur F. Schalk, of Ohio State University, entitled "Points of Interest to Practicing Veterinarians Gathered from Large Animal Clinics, Field Survey and Experimental Work." In this talk Dr. Schalk drew from his rich and extensive experience, and added considerably to the interest of the Conference for the men connected with large-animal practice. This meeting was concluded with pictures of the U. S. Army Cavalry Drill.

Thursday morning dawned bright and fair and the morning session, which was directed by Dr. E. L. Stubbs, of the Pathology Department, was attended by a capacity crowd for Pearson Hall. The first speaker, Dr. Louis A. Klein, professor of veterinary hygiene, caused a stir of interest when he delivered his paper entitled, "Developments in Milk Hygiene." Immediately followed a discourse on "Curd Hardness of Raw and Pasteurized Milk of Philadelphia, and Comparison of the Minnesota and Other Alkaline Reagents with the Babcock Test," which was delivered by Dr. H. C. Campbell of the Department of Bacteriology. These two papers contributed materially to the general knowledge of the latest advances in the dairy industry with its natural problems. Next came a most interesting paper delivered by Dr. H. M. Martin, of the Department of Pathology and Parasitology, discussing the various parasites of the equine subject, and finally, as the last lecture before going to lunch, Dr. W. J. Lentz discussed the more important diseases of the cat, drawing from his rich experience as Director of the Small Animal Clinic.

The final session, held Thursday afternoon, was one of out-

standing scientific interest. With Dr. H. M. Martin in the chair, the meeting opened with greetings from Dr. Thomas S. Gates, president of the University. Dr. R. E. Shope, of the Rockefeller Institute, outlined his meritorious work on swine influenza, and following this Dr. E. L. Stubbs discussed "Leucosis in Chickens," outlining the work done at the University on this unusual disease. Then followed a discussion of poultry practice from various angles by Dr. R. O. Biltz, of the Pennsylvania Bureau of Animal Industry.

The several sessions were well attended and we feel that the interest shown in the Conference is an indication of the spirit which prevails among the members of our profession in spite of the trying economic conditions confronting us. This spirit, we are certain, will carry the profession through to the new era.

A. H. CRAIGE, *Reporter*.

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### CALIFORNIA VETERINARY CONFERENCE

The annual California Veterinary Conference was held at University Farm, Davis, January 4-7, 1933, under the auspices of the Division of Veterinary Science of the University of California.

Some 120 California veterinarians and out-of-state guests attended the four-day meeting which has always proven to be a real attraction, and to which the profession, in all its various branches, looks forward every year. Dr. H. E. Kingman, of Colorado State College, and Dr. Edward Records, of the University of Nevada, were the special lecturers of the Conference. Interesting papers were read at the various sessions by Drs. K. F. Meyer, Director of the Hooper Foundation; C. D. Leake, professor of pharmacology, Medical School, University of California; Geo. H. Hart, professor of animal husbandry; Harold H. Cole, assistant professor of animal husbandry; C. M. Haring, J. Traum, J. R. Beach, Chas. J. Parshall, W. H. Boynton, R. E. Lubbehusen and K. G. McKay, of the Division of Veterinary Science, College of Agriculture, University of California. The State Department of Agriculture was well represented by Drs. H. P. Bonnikson, J. J. King and M. E. McDonald, and the Los Angeles County Live Stock Inspection Department by Dr. L. M. Hurt.

Among the chief attractions of the Conference, both for the small-animal and general practitioners, were a discussion of surgical technic and surgical demonstrations by Dr. H. H. Searles, professor of surgery, and associates, of the Medical School of the University of California. This was the second occasion when,

through the Veterinary Conference and by the same men, California veterinarians have been given an opportunity to see surgery performed on animals by skilled hands and in the same manner as in human practice.

The California State Veterinary Medical Association met at an executive session on the evening of January 5. Friday evening, January 6, a banquet was held at Hotel Woodland, Woodland, Calif. An informal musical program was arranged by Dr. Cliff D. Carpenter, of Los Angeles, who led the members in a group of popular songs. Dr. W. L. Curtis, president of the Association, who was also acting as toastmaster for the evening, called on Dr. Earl E. Wegner, Dean of the Veterinary Division of Washington State College, who addressed the audience briefly. The principal speaker of the evening was Dr. Joseph M. Arburua, of San Francisco, who gave a carefully prepared and a very comprehensive sketch of "The Early History of Veterinary Medicine in the United States." The speaker presented a number of relics in the form of old and rare books, among which was one published as far back as 1806, by Paul Jewett, of Rawley, Mass., entitled "New-England Farrier or a Compendium of Farriery." The presentation of Dr. Arburua's subject was such that those who had the good fortune of hearing him will remember it for a long time to come.

GEO. M. SIMMONS, *Secretary.*

### LONG ISLAND VETERINARY MEDICAL ASSOCIATION

The regular monthly meeting of the Long Island Veterinary Medical Association was held at Canoe Inn at Hampton Bay, January 5, 1933.

Dr. J. F. DeVine, of Goshen, N. Y., gave a very interesting talk on hemorrhagic septicemia in cattle, lead arsenite poisoning in cattle, and enzoötic chorea in Irish wolfhound puppies. caused by a streptococcic infection of the brain. A rising vote of thanks was extended Dr. DeVine for his interesting talk.

The following were elected as officers: President, Dr. W. W. Bennett, East Hampton; vice-president, Dr. Chas. S. Chase, Bay Shore; secretary-treasurer, Dr. Oscar Glueck, Blue Point.

The Association voted to hold the next meeting on April 6, 1933, at Bay Shore, and the Program Committee is planning a clinic in conjunction with the meeting.

OSCAR GLUECK, *Secretary.*

### NORTH CAROLINA STATE VETERINARY MEDICAL ASSOCIATION

According to information received from Dr. J. H. Brown, secretary-treasurer of the North Carolina State Veterinary Medical Association, that organization held a business meeting recently and, among other business transacted, the following resolution was adopted:

WHEREAS, It has come forcibly to the attention of the North Carolina State Veterinary Medical Association that certain county agents operating under the Smith-Lever Act and certain teachers of agriculture operating under the Smith-Hughes Act, both being paid out of state and federal funds, are attempting to give veterinary service to owners of live stock with increasing frequency, a purely personal service, and in direct violation of the spirit and letter of the federal law covering, and

WHEREAS, These men are not qualified by training or experience to perform proper veterinary service; numerous instances have come to our attention recently in which these said agents and teachers have made grossly incorrect diagnoses and used improper treatments, resulting in the death of many animals and in the spread of infectious diseases; and they have otherwise demonstrated their incompetence, depending upon their prestige as state and federal employees to overcome their lack of veterinary training, taking advantage of the agricultural depression to perpetuate their jobs by putting over a plan of free (?) service on an unsuspecting public which, through taxation, is paying their salaries, a plan that appears to be approved by those in charge of the work, and

WHEREAS, Many members of this Association are engaged in the private practice of veterinary medicine, a practice that is essential to the welfare of our live stock industry and encouraged by state and federal governments; these veterinary practitioners have properly equipped themselves at great expense; have complied with all the laws pertaining to veterinary practice; have paid a privilege tax to so practice and are paying taxes to both state and nation to help pay the salaries and expenses of these men; and do not object to these tax-paid employees performing emergency veterinary service, but consider it most unfair competition for them to hold themselves out as qualified to do general veterinary work, leading the uninformed live stock owner to believe that he is getting something for nothing, and resulting in the unnecessary death of many animals, in the spread of animal diseases and in depriving the practicing veterinarian of work which he properly is equipped to do and to which he justly is entitled, therefore, be it

*Resolved*, That this Association go on record as opposing this incompetent and unfair service performed by these tax-paid agents and teachers, in direct violation of the laws covering, and urge its discontinuance, and be it further

*Resolved*, That a copy of this resolution be sent to the Governor of North Carolina, to each of the Senators and Representatives in Congress, to Hon. Jos. B. Shannon, Chairman of the Shannon Congressional Committee, and a copy to the editor of the JOURNAL OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION with the request that same be published.

# NECROLOGY



## DANIEL D. LE FEVRE

Dr. Daniel D. LeFevre, of Newark, N. J., died suddenly on September 28, 1932. He was born at Bartlett, Tenn., December 15, 1873, and was a graduate of the New York State Veterinary College at Cornell University, class of 1903. He was a resident of Newark, N. J., during most of his lifetime, and was active in veterinary organizations as well as in the affairs of his community.

Dr. LeFevre joined the A. V. M. A. in 1928. He was a member of the New York State Veterinary Medical Society and the Geneseo Valley Veterinary Medical Society. He served the latter as president in 1928.

J. G. W.

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## HENRY O. KELPE

Dr. Henry O. Kelpe died at his home in Omaha, Neb., October 21, 1932, after an illness dating back several years. Born in Centain, Mo., July 14, 1877, Dr. Kelpe received his veterinary education at the Kansas City Veterinary College. He was graduated with the class of 1909 and almost immediately entered the service of the U. S. Bureau of Animal Industry. Before being stationed at Omaha, on stockyards inspection, he was located at Albuquerque, N. M.

Dr. Kelpe joined the A. V. M. A. in 1910. He was a member of the National Association of B. A. I. Veterinarians. He is survived by his widow, two sons and two daughters.

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## GEORGE PERRY LYNCH

Dr. George P. Lynch, of Montevideo, Minn., died October 24, 1932. Death was due to heart failure while on a hunting trip.

Born in Des Moines, Iowa, June 13, 1881, he had been a resident of Montevideo for the past twenty-one years. He was a graduate of the Grand Rapids Veterinary College, class of 1916.

Dr. Lynch is survived by his widow (née Anna Larson), three daughters, his mother, two brothers and four sisters.

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**LYMAN D. BROWN**

Dr. Lyman D. Brown, of Hamilton, Mo., died at his home, November 27, 1932. He had been in poor health for several years.

Born at McArthur, Ohio, August 25, 1855, Dr. Brown attended the Chicago Veterinary College. Following his graduation in 1897, he conducted a general practice at Hamilton, Mo., until about 1908, when he entered the service of the Missouri Department of Agriculture as a field veterinarian. He continued in this work until 1922, when impaired health compelled him to give it up.

Dr. Brown joined the A. V. M. A. in 1904 and resigned in 1931. He served as Resident Secretary for Missouri, 1922-23. He was a member of the Missouri State Veterinary Medical Association. Surviving Dr. Brown are his widow and two daughters.

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**HARRY BOSLEY**

Dr. Harry Bosley, of Washington, D. C., died December 13, 1932, after a short illness due to renal complications which followed an operation on the prostate gland.

Born in Baltimore County, Md., September 18, 1866, Dr. Bosley attended grade schools and later entered the United States College of Veterinary Surgeons in Washington, D. C. He was graduated in 1903 and held the chairs of anatomy and dentistry in the institution for a number of years, while conducting a general practice at the same time. For the past fifteen years, his son, Dr. Milton A. Bosley (U. S. '17) had been associated with him.

Dr. Bosley joined the A.V.M.A. in 1918. He is survived by his widow and three sons.

H. E. M.

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**GEORGE D. WOOD**

Dr. George D. Wood, of Springfield, Vt., died on December 22, 1932, after a brief illness. He was born at Strafford, Vt., January 27, 1869. He attended common schools and Thetford Academy before entering the Ontario Veterinary College. He finished the two-year course in 1908, returned the following fall for another year of study, and was graduated again with the class of 1909. He was engaged in general practice first at Hartland, Vt., and later at Springfield.

Dr. Wood joined the A. V. M. A. in 1924. He was a member of the Vermont Veterinary Medical Association. His widow survives him.

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**HERBERT H. BROWN**

Dr. Herbert H. Brown, of Saint Louis, Mo., died at his home, December 27, 1932, of heart disease, after a lingering illness.

Born at Rich Hill, Mo., February 11, 1876, he attended grade and high schools before entering the customs service in 1904. He later held positions in the Post Office Department (1909-10) and in the War Department at Washington (1910-16). While holding the latter position, Dr. Brown attended the Veterinary Department of George Washington University, in Washington. He was graduated with honors in 1916 and immediately entered the service of the U. S. Bureau of Animal Industry. He was assigned to meat inspection at National Stock Yards, Ill., where he remained for six months. He was then transferred to Saint Louis, where he remained until his death.

Dr. Brown joined the A. V. M. A. in 1921. He was a member of the National Association of B. A. I. Veterinarians. He is survived by his widow.

J. S. K.

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**CHARLES R. REY**

Dr. Charles R. Rey, of Tulare, Calif., was killed in an automobile accident, near Madera, January 7, 1933, while returning home from Davis, Calif., where he attended the annual California Veterinary Conference at University Farm, January 4-7.

Born near Hanford, Calif., in 1878, Dr. Rey was a graduate of the San Francisco Veterinary College, class of 1913, and had been in practice at Tulare for the past seventeen years.

Dr. Rey joined the A. V. M. A. in 1913. He was a member also of the California State Veterinary Medical Association and the San Joaquin Valley Veterinary Medical Association. He is survived by his widow, three sisters and two brothers.

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**OSWALD HOOD GRAHAM**

Dr. O. H. Graham, of Tarboro, N. C., died in a local hospital, after a brief illness from a heart attack, December 28, 1932. He was 45 years of age.

Born in Scranton, S. C., Dr. Graham was graduated from Clemson College in 1912 with the degree B. S. In 1914 he received his veterinary degree from Ohio State University. He practiced at Clinton, N. C., for a short time after graduation and was appointed Assistant State Veterinarian of North Carolina, becoming State Veterinarian August 1, 1917, which position he

held until November 1, 1918, at which time he resigned to enter military service. Upon his discharge from the Army in 1919, he located in Tarboro. He developed a splendid practice in that territory and was very successful as a practitioner.

Dr. Graham joined the A. V. M. A. in 1915. He was a member of the North Carolina Veterinary Medical Association and also a member of the North Carolina State Veterinary Medical Examining Board.

W. M.

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### JOHN J. HAYES

Dr. John J. Hayes died at San Diego, Calif., January 9, 1933. He was a graduate of the American Veterinary College, class of 1894, and conducted a general practice in New York City until his retirement about ten years ago. The deceased was a cousin of Dr. John J. Hayes (N. Y.-Amer. '00), formerly of Chicago and now located in New York City.

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### SAMUEL R. HOWARD

Dr. Samuel R. Howard, of Hillsboro, Ohio, died at his home, January 11, 1933, from heart trouble. He had been ill for the past six months and suffered a stroke of apoplexy several days before his death.

Born in Circleville, Ohio, June 7, 1861, Dr. Howard studied veterinary medicine at the Ontario Veterinary College. He located at Hillsboro immediately following his graduation in 1889, and practiced there for 42 years. He was a member of the Ohio State Veterinary Medical Association, and was placed on the Honor Roll of that organization in 1917, upon the completion of twenty-five years of membership.

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### WALTER LANGTRY

Dr. Walter Langtry died of a heart attack at the home of relatives in Houston, Tex., January 14, 1933, at the age of 77. He had been in Texas for about a year, his previous residence having been in Fort Wayne, Ind., where he practiced from the time of his graduation from the Ontario Veterinary College, in 1877. He was a native of Canada.

Dr. Langtry joined the A. V. M. A. in 1911. He was active in fraternal and civic affairs in Fort Wayne for many years. Surviving him are a son and a daughter, as well as brothers and sisters in Canada.

**JOHN FRANKLIN BLINZLEY**

Dr. John F. Blinzley, of Bellevue, Ohio, died at his home, January 24, 1933, after an illness of several months. Born near Pontiac, Ohio, June 24, 1861, Dr. Blinzley attended local schools and the Ontario Veterinary College. Following his graduation in 1895, he located at Bellevue and engaged in practice there until a few weeks before his death. He is survived by his widow, one daughter, two stepchildren and three brothers.

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Samuel W. Garrett, of Birmingham, Ala., a junior student in the College of Veterinary Medicine, Ohio State University, committed suicide by swallowing poison, January 19, 1933. Dr. Thomas W. Garrett (O. S. U. '32), now in California, is a brother of the deceased.

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Our sympathy goes out to Dr. Ray Hoeftling, of Austin, Minn., in the death of his mother, Mrs. Mary J. Hoeftling, at Austin, Minn., January 20, 1933, at the age of 71 years, after an illness of about six months.

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**PERSONALS**

DR. F. H. McCLOUGHRY (Gr. Rap. '10) has located at Imlay City, Mich., for general practice.

DR. GUY L. O'HARRA (K. C. V. C '18), formerly of Newman, Calif., is now located at Los Baños, Calif.

DR. DANIEL DECAMP (K. S. C. '29) has been transferred again, this time from Fort Wayne, Ind., to Oregon, Mo.

DR. CARL E. CHASE (O. S. U. '28) has opened the Coniston Pet Hospital, at 1153 Hanover St., Manchester, N. H.

DR. FRED W. MEIER (Mich. '32) has been pursuing postgraduate work at the Tierärztliche Hochschule, Berlin, Germany.

DR. J. E. HICKEY (Chi. '13), of Juneau, Wis., assumed office as register of deeds of Dodge County the first of the year.

DR. A. M. STROM (Ind. '21), formerly of Ortonville, Minn., has been in Arizona for several years trying to regain his health.

DR. J. A. CAMPBELL (Ont. '00) is a member of the Board of Governors of the Toronto Humane Society, Toronto, Ontario, Canada.

DR. W. D. MANDEVILLE (Chi. '18), of Waukesha, Wis., has taken over the practice of Dr. J. E. Hickey (Chi. '13), at Juneau, Wis.

DR. FOREST DAVIS (Chi. '12), of Moulton, Iowa, was elected to the Iowa legislature at the November election. He represents Appanoose County.

DR. FREDERICK P. RUHL (Amer. '85), of Milford, Del., returned to his home about the middle of January after spending a month's vacation in Florida.

DR. ALANSON L. ALBRIGHT (McK. '03), of Cayuga, Ind., is a member of the Indiana State Senate, representing Fountain, Vermilion and Warren counties.

DR. L. R. RICHARDSON (O. S. U. '31), of Kent, Ohio, has removed to Ravenna, Ohio, where he has taken over the practice of Dr. R. N. Owen (O. S. U. '23).

DR. HARRY L. McEWAN (Chi. '08), of Elburn, Ill., recently purchased the Kane County Bank Building in that city and will use it for his office and laboratory.

DR. C. L. BUTLER (K. S. C. '29), formerly with the American Society for the Prevention of Cruelty to Animals, New York City, is now located at Easton, Md.

DR. W. C. HOLTZ (Chi. '07), of Chicago, Ill., was critically ill the past month, from influenza complicated with a heart affection. He was up and around at last reports.

DR. J. L. WRIGHT (U. P. '32), who has been assisting Dr. O. M. Norton (U. P. '01) at Greenville, Miss., since graduation, has located at Chatsworth, Ga., for general practice.

DR. L. L. BECHTOL (O. S. U. '32), of Napoleon, Ohio, has taken over the practice of the late Dr. E. C. Wisman (Ont. '95), at Bryan, Ohio, whose death was reported in the January issue of the JOURNAL.

DR. E. E. HAMANN (Mich. '31) and Dr. J. H. CAMPBELL (Mich. '31), both of Saint Johns, Mich., have formed a partnership and will conduct a practice and hospital at 2601 East Michigan Avenue, Lansing, Mich.

DR. S. T. MICHAEL (Iowa '26) resigned his position in the Division of Veterinary Science, University of California, Berkeley, November 1, 1932, and is now in charge of the S. P. C. A. Hospital in San Francisco.

DR. N. S. MAYO (Chi. '89), of Highland Park, Ill., accompanied by Mrs. Mayo, was a Florida visitor the past month. They were last heard from at Mount Dora, and expected to visit Cuba before returning home.

DR. RAY HOEFELING (Cin. '11), of Austin, Minn., returned to his B. A. I. station on November 16, 1932, after an absence of over eight months, due to an attack of neurofibrositis, diagnosed at the Mayo Clinic, Rochester, Minn.

DR. GEORGE H. BERNIS (Col. '79), of Brooklyn, N. Y., who met with an accident in December, as reported in the January issue of the JOURNAL, continues to improve, but very slowly, according to a recent letter from his daughter, Miss Nellie C. Bernis.

DR. L. J. GALLAGHER (Ont. '17), who was employed by the Wisconsin Department of Agriculture and Markets for the past eight years, has entered private practice at 1000 Eighth Avenue West, Ashland, Wis. Previous to entering state employ Dr. Gallagher practiced at Elroy, Wis.

## TWELFTH INTERNATIONAL VETERINARY CONGRESS

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